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THE

# AMERICAN YEAR-BOOK

OF

# MEDICINE AND SURGERY

BEING

A Yearly Digest of Scientific Progress and Authoritative
Opinion in all Branches of Medicine and Surgery,
drawn from Journals, Monographs, and Text-
Books, of the Leading American and Foreign
Authors and Investigators

COLLECTED AND ARRANGED

WITH CRITICAL EDITORIAL COMMENTS

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UNDER THE GENERAL EDITORIAL CHARGE OF

GEORGE M. GOULD, M. D.

## MEDICINE

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1901

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# PREFACE.

THE experiment made last year in issuing this work in two volumes has proved very acceptable to subscribers and is continued.

The increased popularity of the YEAR-BOOK is gratifying to the editorial department.

The single change in editorship is the association of the scholarly pathologist Dr. Aloysius O. J. Kelly with Dr. Riesman in the section on Pathology.

GEO. M. GOULD.

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# GENERAL MEDICINE.

BY ALFRED STENGEL, M. D., AND D. L. EDSALL, M. D.,
OF PHILADELPHIA.

## INFECTIOUS DISEASES.

### GENERAL CONSIDERATIONS CONCERNING INFECTIOUS DISEASES.

E. Walger [1] in presenting some theoretic considerations concerning the importance of the **anatomic changes in acute infectious diseases** strongly insists that these alterations should not be looked upon as constituting the disease, but as being the expression of the attempt of the system to rid itself of the toxins, and he especially insists that the truth of this view may be seen in certain cases of pneumonia. He records the case of a man of 29 who was taken ill with an acute lobar pneumonia. He was at once injected with 8 cc. of serum taken from a man convalescing from pneumonia. There was no change in the condition on the next day, but on the second day practically all the subjective symptoms had disappeared, and the temperature soon reached normal and remained so, the man's general condition being outwardly practically that of health. Nevertheless, the lung proceeded to consolidation, which ran its usual course, and resulted in entire resolution. This Walger states is good evidence that the exudation in the lung is merely the method used by the system to rid itself of the toxins. [While we may agree with the author's general proposition, the proof adduced seems rather unsatisfactory.]

W. F. Phillips,[2] in discussing **clinical thermometry,** insists that uniform methods of taking the temperature should be adopted; until this becomes general, records should be accompanied by some sign showing whether the temperature was taken in the mouth, axilla, or rectum. He recommends the axilla, and that the bulb of the thermometer should be of the same diameter as the stem, so that the entire bulb will come in contact with the skin of the armpit when the temperature is being taken. The bulb should be placed well up in the apex of the axilla. The mean temperature of normal individuals he finds to be 97.4° F. and the normal range 97° to 98.4° F.

E. v. Czyhlarz [3] studied the **effects of high temperature upon the animal organism** by puncturing the medullas of 10 rabbits. He examined the kidneys, hearts, muscles, and livers of these animals, particularly to see whether there was fatty degeneration or parenchymatous degeneration. In no instance was either found. He also reports that the blood was entirely normal, and that the specific gravity in particular was investigated, and found to be normal. The latter is of interest, in view of the teaching of some, that there is a hydremia in fever.

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[1] Centralbl. f. innere Med., Dec. 10, 1899.
[2] Brit. Med. Jour., Oct. 28, 1899.
[3] Centralbl. f. innere Med., July 22, 1899.

A. Lode and A. Durig[1] find that animals, dogs especially, show a decrease in the **influence of cold baths** upon the temperature when the cold baths are repeated. The fall in the temperature on the first day, for instance, was 42° F.; on the sixth and seventh days the fall had decreased to 32° F. The cause of this declining influence of cold was not increase in metabolism and the consequent increased heat production, but was lessened heat-elimination. It was an evidence of adaptation to new conditions.

E. B. Holbert[2] describes a curious case of a man of 36 who was thought during life to have probable sclerosis of the brain, but autopsy disclosed no lesion. The case is reported chiefly because of the marked **hypothermia,** which is said to have reached 81.6° F. when taken in the rectum. After treatment with hot baths the temperature is said to have risen as much as 19.5° F. in 18 hours, but fell again, and remained subnormal until a short time before death, when it rose to 104° F.

A. MacFayden and S. Roland[3] report their experiments concerning the **influence of liquid air** upon bacteria. They exposed a series of organisms for 20 hours to liquid air, the temperature being from —183° C. to —192° C., without observing any changes in the virulence of the organisms or in their morphology. The typhoid, colon, diphtheria, proteus vulgaris, lactic acid, and anthrax bacilli, the spirillum of cholera, the staphylococcus, and other organisms were used. Later[4] exposure for 7 days was carried out, the organisms being placed in small hermetically sealed tubes and completely immersed in the liquid air. In spite of the enormous mechanical strain to which the organisms were thus subjected, and exposure to a temperature of —190° C. for 7 days, no alteration was seen in their structure and there was no change observed in their virulence except that they grew a trifle more slowly.

W. Wecksberg[5] gives a very interesting report of some experimental work relating to the **influence of chemical counterirritants upon inflammation.** Experiments were carried out by shaving the skin of dogs and applying counterirritants, chiefly iodin, croton oil, and mustard. The result of the work was to show the presence of decided edema and infiltration, not only of the skin, but of the subcutaneous tissue, and often of the muscles beneath; this was most marked after the use of croton oil. There was decided hyperemia of the vessels in the superficial area, and to produce such a hyperemia Wecksberg believes that there would necessarily be compensatory anemia in the surrounding regions. But the more important point in his mind is the edema, which causes direct compression upon the blood-vessels of the underlying parts, and therefore would cause anemia in these regions. He then investigated the actual influence of counterirritants upon local inflammatory processes by producing first aseptic abscesses by injecting turpentine, afterward applying iodin around the areas in some cases, and using others as controls. The iodin regularly influenced the subsequent course favorably, and, if irritation had not been too severe, prevented the formation of abscesses, while in the controls abscesses always occurred. Similar results were obtained after injecting staphylococcus cultures. If the edema causes the chief changes, it seems strange at first that the application of counterirritants relieves pain, as it would be thought that pressure of the edema might cause pain. But Wecksberg explains this by the statement that inflammatory edemas are accompanied by hyperemia, while the edema produced by the local counterirritants is an anemic edema; also the edema produced by the latter occurs very rapidly and is likely to cause such rapid and severe pressure as to obtund the sensitiveness of the nerves.

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[1] Münch. med. Woch., Jan. 23, 1900.

[2] Lancet, Jan. 13, 1900.

[3] Lancet, Mar. 24, 1900.

[4] Lancet, April 21, 1900.

[5] Zeit. f. klin. Med., Bd. XXXVII, Hefte 3 u. 4.

Weisbecker[1] reports the use of the **serum of convalescents** from measles, scarlet fever, typhoid fever, pneumonia, and diphtheria in the treatment of these diseases, and especially discusses the method of obtaining and using the serum. In the first place, the patient from whom it is obtained must be decidedly ill, mild cases not being of value. In pneumonia, serum should be taken about the seventh to the ninth day, also the patients should not have been treated by any active medication, and antitoxins should never have been used. He disapproves of the use of anodynes, expectorants, etc., as well as antipyretic measures or drugs in the subject of the venesection. In typhoid fever he takes blood on the fourth or fifth day of convalescence. The blood he withdraws into glass vessels, taking about 250 cc. on the average from adults, though more may be taken. It is stood aside, and after 48 hours the serum is withdrawn and placed in glass vessels of 10 cc. to 15 cc. capacity. He does not consider it necessary to add an antiseptic, but it is safer to add a few drops of a 0.5 % to 1 % solution of carbolic acid. The dose is 10 cc. in adults, 5 cc. in children. The injection usually causes some pain. The use of the serum he considers indicated in all cases except those that are very mild, and it is particularly to be recommended in old persons. It should be given very early in the case. One injection is often enough, but if the symptoms become worse afterward, he performs venesection, and then gives another smaller injection.

H. Davidsohn[2] in order **to maintain the heat of cataplasms** covers them with an apparatus consisting essentially of a rubber tubing placed upon linen. Hot water is passed through the tube constantly. A mattress similar to this has also been constructed which helps to maintain the body-heat, or may be used for applying heat to the back.

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[1] Münch. med. Woch., Aug. 9, 1899.

[2] Berl. klin. Woch., Jan. 19, 1900.

## TYPHOID FEVER.

Antony [1] describes an interesting observation concerning the **etiology of typhoid fever.** Two regiments which were lodged in the same barracks were served from the same **water-supply.** In one regiment there was a number of cases of typhoid fever, while in the other none occurred. The explanation of this proved to be that in one regiment the water was drunk without boiling, while the soldiers in the other regiment for months had taken nothing but tea—the water, of course, having been boiled. In discussion Chantemesse suggested that the occurrence of typhoid fever in only a part of a series of persons apparently exposed to the disease might be explained by the fact that, by their presence, **other micro-organisms favor infection** with the typhoid bacillus. Certain persons contract typhoid fever without drinking water which is definitely infected. This he attributes to the fact that typhoid bacilli are taken into the intestine frequently or remain there for a long time without being able to produce disease, but if water infected with other organisms is drunk, the presence of these other organisms is sufficient to precipitate a typhoid infection. [The view that preliminary intestinal infection of various parts predisposes to typhoid infection seems likely from certain observations, and may indeed be a factor of great importance. It would be interesting to investigate this subject experimentally were animal infection more certainly established for this disease.]

F. S. Crum,[2] in studying the **mortality** of typhoid fever **in 24 American cities,** emphasizes the unpleasant fact that throughout this country typhoid fever mortality is exceedingly high. It is noteworthy, however, that there has been a marked reduction in the death-rate during the last 10 years in most of the cities, New Orleans alone showing an increased mortality. The reduction in the mortality in Newark from 99 per 100,000 to as low as 14 per 100,000, is very instructive, since the city **water-supply** was altered just at the time that that reduction occurred. Securing water from a high, thinly settled district at a distance was coincident with a drop within 2 years from 99 per 100,000 to 22 per 100,000. A similar improvement in Chicago was also coincident with an improvement in the death-list from typhoid. [In considering statistics it is always to be remembered that diagnosis has become more accurate. Many fatal cases of typhoid were formerly classed as malaria. This mistake is far less frequent now. On the other hand, however, it must be acknowledged that the milder cases that are now recognized, and that tend to lower mortality, were not correctly diagnosed in former times.]

Genersich [3] reports an epidemic of typhoid fever in a Hungarian town in which 209 cases occurred, 28 of which were fatal. The **drinking-water** used by these people was taken from two cisterns, and a bacteriologic examination of the water showed the presence of the **typhoid bacillus.**

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[1] Gaz. des Hôp., July 7, 1899.

[2] Med. Rec., Aug. 12, 1899.

[3] Centralbl. f. Bakt. Parasit. u. Infect. Krankh., 1900, Nos. 7 and 8.

C. C. Hubbard[1] describes an epidemic of typhoid fever which occurred in Worthville, N. C., a village of about 350 inhabitants. There were 63 certain cases and a number of others which were doubtful. **Infection** in about half the cases apparently occurred **from one well,** and the epidemic came on at a time of the year when water was very low and when a number of the wells of the village had gone dry. The death-rate was about 10%.

P. E. Archinard[2] notes that earlier clinicians in New Orleans considered **typhoid fever uncommon,** but that the disease is now frequently seen. He believes that this is due to actual increase in the disease, because, chiefly, of increase in population, the bad drainage obtaining in the city, and especially the prevalent custom of using rain-water for domestic purposes.

J. C. Tresh and E. R. Walter[3] describe an outbreak of typhoid fever which was probably due to **infection from eating cockles,** since the water-supply seemed to be uninfected and there was no evidence of infection of the milk-supply. The sewage arrangements were good and there was no pollution of the soil, so that these factors seemed to be excluded, and the cockles seemed to be the cause, since practically all those who were attacked had eaten them, and they were obtained from a source which was polluted by sewage. Almost all of those who were taken ill were recent arrivals in the place where the outbreak occurred.

W. H. Park[4] reports his discovery of the **typhoid bacillus** in the feces in 40% of 104 cases. One epidemic of typhoid fever which he reports was due to contamination of ice; another was the result of eating raw cockles, the water in which the latter had grown having been infected by sewage. He believes that it is possible to immunize against typhoid fever by injecting serum from an immunized horse or by injecting agar cultures of the typhoid bacillus which have been heated up to 55° C. or to which $\frac{1}{2}$% carbolic acid has been added. The Widal reaction, he finds, occurs in about 95% of cases. If it is found in a dilution of 1 : 10, higher dilutions should be investigated.

C. A. Cameron[5] describes a series of small epidemics of typhoid fever due to **infection from the milk** which was served to all the people who became ill. It was learned that cases of typhoid fever had occurred at the dairy from which the milk was supplied, and the woman who had nursed the patient with typhoid fever also attended to the business of the dairy. As a result there were in one institution 32 cases of typhoid, in another 6, in another 4, in another 14; besides this, there was a series of cases in private houses which had been supplied from the same dairy.

V. C. Vaughan[6] contributes a paper upon **typhoid fever** as it appeared **among the soldiers** in the war between the United States and Spain. Vaughan's paper is the result of an investigation by the Government Board, of which he was a member. He notes first that the army surgeons' diagnosis of typhoid fever was in many cases very unreliable. Most cases of fever were diagnosed malaria; but so far as can be determined, malaria was rare among the troops, while typhoid fever was extremely common. The infection in the camps was brought by soldiers from their homes. One fact that is considered of great importance in having caused the spread of the disease is that in many instances the surgeons unwisely suppressed the diagnosis in order to prevent alarm. The disease did not come from drinking-water, since residents of nearby towns used the same water and did not become infected, while the soldiers did. The general infection was believed to have been due chiefly to the method of depositing excrements in pits. The disease was believed to have been carried by flies. There was no evidence of infection through milk. The Board of Investigation insists that it is of the utmost importance to sterilize completely all fecal matter, and recommends for future use a galvanized iron trough, which should contain a saturated solution of milk of lime and should be emptied daily by odorless excavation. Each company should have such a trough. The Board also insists that medical officers of the army should have special training in military hygiene. The mortality from typhoid is said to have been between 4% and 7%. Considering the effect of changing the location of the camp, the Board decided that mere change in location, even if the sick are left behind, does not rid the camp of typhoid. It is necessary to disinfect the belongings of the camp. If, however, the move be made before there is wide-spread infection, the disease may disappear. A sea voyage lasting some days or more seems to be of decided use in ridding the command of typhoid fever, though this did not seem to be of marked effect if there was already a wide-spread infection among the men.

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[1] Med. News, Dec. 30, 1899.

[2] Phila. Med. Jour., Mar. 31, 1900.

[3] Brit. Med. Jour., Dec. 16, 1899.

[4] Med. News, Dec. 16, 1899.

[5] Dublin J. M. Sc., Nov., 1899.

[6] Am. Jour. Med. Sci., July, 1899.

H. Peck[1] discusses 206 cases of typhoid fever which he has investigated in connection with the question of **sick-room infection.** He decides that in 28 of these cases the patient probably received the infection in the sick-room, and considers that such a manner of infection is much more common than is usually believed, and that the danger of such infection should be recognized and guarded against. Infection in the sick-room is particularly likely to occur in the small houses of the poor, where people are crowded together. [We can confirm the author's views from our own experience, and have become impressed with the opinion that the profession does not sufficiently recognize the danger of infection in the sick-room. The friends of patients are allowed to become careless through the well-meant assurances of the physician that typhoid fever is not contagious. This statement, without explanation of its limitations, naturally leads to carelessness.]

**Pathology.**—R. Lepine and B. Lyonnet[2] report some experiments concerning **typhoid infection in the dog.** They first fed the animal on bouillon cultures of typhoid fever, without the production of any lesions or of agglutinative power in the blood-serum. The injection of virulent cultures into the intestine, and ligation of the intestine above

[1] Brit. Med. Jour., Sept. 2, 1899.

[2] Rev. de méd., Aug. 9, 1899.

and below the site of injection, caused no result except enlargement of the mesenteric glands. Afterward a loop of intestine was isolated by Thiery's method, and bacilli were injected into the isolated loop. This induced marked agglutinative action of the blood-serum, peritonitis, and intestinal lesions resembling those seen in man; bacilli were recovered from the mesenteric glands. Injection of cultures into the mesenteric vein resulted as follows: The bacilli were found in the spleen 8 times, in 1 case the result was doubtful, in 1 case negative; 8 positive results were obtained from the examination of the liver, only 2 positive results from the kidneys, 1 positive and 1 doubtful from the lung, 3 negative and 1 positive from the heart blood. Injections of cultures underneath the intestinal peritoneum resulted in finding typhoid bacilli in the spleen regularly; in 5 out of 7 experiments it was found in the liver and 3 out of 5 times in the bone-marrow, and not in any instance in the kidney. Injection of bacilli into a vein of the larger circulation was done 7 times and produced some appearance of general infection in all cases. Injections into the trachea caused death in 2 cases, probably from toxemia; in 3 cases there was typical red hepatization of the lungs, but there was no fever, and the animals probably died from toxemia.

W. Ophüls[1] describes a case of **typhoid infection without intestinal lesions** which occurred in a man of 24 who was admitted to the hospital in a semiconscious condition. The diazo and Widal reactions were positive. The patient died a week later. The necropsy showed enlargement and softening of the spleen and lymph-glands, necroses in the liver, and multiple hemorrhages in the mucous membranes of the genito-urinary tract; bacteriologic examination showed in the spleen large numbers of bacilli which had the characteristics of typhoid bacilli. Ophüls believes that in a certain number of these cases which are apparently without intestinal lesions infection has taken place in the usual way, through the intestinal mucous membrane, but there has been no ulceration of Peyer's patches and the swelling has disappeared. As proof of this, he notes that often in even those cases there is evident irritation of the intestines, and that in many instances death takes place late in the disease when the lesions of the intestines might have disappeared entirely if they had not ulcerated. [The existence of diarrhea and other signs of intestinal irritation in typhoid fever by no means proves that there has been any lesion in the intestine. There is little doubt that the diarrhea is often due far more to the general toxemia than to the local lesions.]

A. McPhedran[2] describes a case which ran the usual course of typhoid fever and presented positive Widal and diazo tests. Death occurred about 40 days after admission to the hospital. The intestines were found free from lesions except inflammation and small hemorrhages, but cultures from the spleen showed the presence of the typhoid bacillus. It was evidently a case of typhoid without lesions of the intestines.

A. J. Lartigau[3] describes 2 cases of typhoid fever with **multiple**

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[1] N. Y. Med. Jour., May 12, 1900.

[2] Canadian Jour. of M. and S., Oct., 1899.

[3] Boston M. and S. Jour., Sept. 7, 1899.

**ulcerations of the vulva and vagina** from which the typhoid bacillus was isolated. The fact that the ulcers were multiple suggested strongly that the origin was in minute bacillary emboli. [We have seen 1 case with similar lesions of the vulva. The ulcers were deep and irregular, and in the assured absence of any form of venereal infection, were regarded as probably due to the existing typhoid infection. They occurred early in the disease when the patient's general condition and circulation were little affected. Unfortunately, no cultures were obtained.]

**Symptomatology.**—W. H. Thomson [1] reports his experience with **368 cases of typhoid fever**—a 10 years' experience. The mortality was 6.8 % ; but if the deaths which occurred during the first week are excluded, the fatalities were but 3 %. Diarrhea was relatively rare, while constipation was common, the infrequency of diarrhea being attributed to the fact that meat-broths were but little used in the diet; Thomson believes that such foods easily produce diarrhea through undergoing fermentation. Tympanites was seen in 20 %, and was a bad sign. Hemorrhage occurred in 7.3 % ; the best treatment was hypodermoclysis of from 8 to 12 ounces of normal salt solution. There were 4 cases of perforation, 2 of which were successfully operated upon. Delirium was rare. Peripheral neuritis was seen 24 times. It was attributed to the foot-drop resulting from the prolonged relaxation of the leg muscles. Phlebitis occurred 7 times and cutaneous abscesses 29 times; the latter appeared in epidemic form in 1894 and were thought to be due to infection from bathing in Croton water. There were 15 % of relapses. In one fatal case intestinal lesions were absent, but there was a severe pelvic myositis, which was thought to be typhoidal. In treatment Thomson uses a mercurial purge every third night for 2 weeks, and gives milk exclusively up to the fourth week. He believes that pepsin and bismuth have great value in improving digestion and thus preventing inflammations of the mouth, throat, middle ear, and parotid glands.

A. A. Smith [2] reports a series of **104 cases of typhoid fever** seen in Bellevue Hospital. There was a history of marked alcoholism in 15 % to 20 %, chilly feelings were noted in about 50 %, diarrhea was present in the same percentage, epistaxis in about 41 %, characteristic spots could not be observed in 20 %, enlarged spleen was absent in 20 cases, the Widal reaction was absent in 10 cases and present in 49, the diazo reaction was present in 44 and absent in 15. There were 8 relapses and 10 deaths. In 5 cases the **plasmodium of malaria** was seen in the blood during convalescence. In 1 case the intestine was aspirated for the relief of tympanites; peritonitis occurred and the patient died. In 3 cases death was due to exhaustion, in 2 to perforation, in 1 to edema of the glottis, and in 2 to hypertrophy.

H. E. Belcher [3] reports a case of typhoid fever which began on July 25th, and in which there was a continuous high temperature until September 15th, when it touched normal. On the 16th the temperature rose again, and did not reach normal finally until November 5th—the **one hundred and fifth day of the disease.** The patient repeatedly passed blood from the bowel. He was in unfavorable hygienic surroundings, and after the last rise of temperature was removed to a healthful house, which probably helped in the termination of the disease.

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[1] Med. Rec., Nov. 11, 1899.

[2] Med. News, Dec. 9, 1899.

[3] Brit. Med. Jour., April 7, 1900.

W. Murrell [1] describes a case of typhoid fever in which the temperature persisted continuously **above the normal for 80 days.** He describes this as an attack of prolonged typhoid fever, and believes that it was not relapse, complication, or post-typhoidal pyrexia.

Etienne [2] describes a case of **ambulatory typhoid fever** without rise of temperature, but in which there was a distinct typhoid state and a marked Widal reaction, the typhoid state persisting for 36 days. In discussion Widal stated that he had never seen apyretic typhoid fever, but that he had often observed at the end of the disease that there was a **dissociation between the febrile symptoms of the disease and the other symptoms.** For instance, he had also repeatedly seen severe general symptoms persist with a typhoid state after the fever had fallen to normal, and had so remained for several days. He had also repeatedly seen the contrary. He suggested that it would be very interesting to follow the course of the reaction of agglutination and the diazo reaction in cases of apyretic typhoid fever. In some systematic observations which he had made he had seen the diazo reaction persist after the fever had fallen in such cases.

P. Remlinger [3] contributes an article on **rubeoliform and scarlatiniform eruptions** in typhoid fever. He reports an interesting series of 12 cases which he has observed in the past 2 years at the Belvédère Hospital; 7 of them were rubeoliform, 1 was scarlatiniform, and 4 were mixed. Taking the 49 observations which Remlinger has collected, one sees that there have been 31 cases of rubeoliform, 14 cases of mixed eruptions, and only 4 of scarlatiniform eruptions. They almost always occur in persons under 25, and show a marked tendency to appear in children and in military services. The eruption is practically always seen on the trunk, but frequently affects the extremities also, and at times the face. The spots are sometimes discrete and sometimes confluent. They are usually very ephemeral, commonly lasting 3 or 4 days; they sometimes leave no trace behind, but quite frequently terminate by desquamation. All 4 scarlatiniform cases desquamated, and desquamation was observed in 4 of the 14 of mixed form. The rubeoliform eruptions are likely to leave for some time a copper-colored stain resembling that seen in syphilis. Recurrences have been seen. There are usually no sensory symptoms. The temperature varies, is often unaffected, often rises, and sometimes falls. General symptoms are usually not marked, but may occur; albuminuria is, however, frequent. There is a striking tendency to epidemics of these eruptions. Hutinel has observed 12 cases in one series of 38 cases of typhoid in children;

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[1] Brit. Med. Jour., June 7, 1900.

[2] Gaz. des. Hôp., Jan. 26, 1900.

[3] Rev. de méd., Feb. 10, 1900.

5 of the 12 died. There also seems to be a contagiousness about the eruptions, and it has repeatedly been observed that they occurred in several patients who occupied the same bed consecutively. There have been no characteristic pathologic and bacteriologic discoveries in connection with the eruptions. In general they **indicate a grave prognosis.** The scarlatiniform eruption, while rare, is usually associated with a grave form of the disease. The rubeoliform and mixed forms usually fall into 2 categories: those which occur during the course of the disease are commonly associated with grave general symptoms, and with a likelihood of fatal issue; those which occur during defervescence are not of grave import, as a rule.

J. M. Da Costa[1] discusses **anomalous eruptions** in typhoid fever and reports cases. He first considers scarlatiniform rash, which may be a uniform eruption over the body or may occur in patches. It is of varying degrees of intensity and commonly lasts about a week; it is not followed by desquamation, is rarely connected with sore throat or albuminuria, and apparently does not influence the temperature. It is usually easy to exclude scarlet fever. The measles-like rash, which is less common, often renders the diagnosis difficult. In some instances it suggests the possibilities of measles, and in others causes the case to resemble typhus. One patient with such a rash was isolated because it was thought that he had typhus. As to measles, this disease and the eruption resembling it are differentiated by the fact that the measles eruption is more crescentic in its arrangement, more decidedly papular, and coarser. It causes itching and is followed by desquamation. These are not observed in the measles-like rash in typhoid fever, and in the latter there are none of the catarrhal symptoms of measles. If measles occurs with typhoid fever, the temperature rises markedly preceding the appearance of the eruption, and remains high as the eruption spreads. This is a very important diagnostic point. Besides the measles and scarlet fever rashes, there is often a mottled appearance of the skin which may resemble typhus. Da Costa thinks that these eruptions are dependent upon the same basis, which is, in his belief, a vasomotor disturbance of the cutaneous nerves due to infection. The nervous element is of much importance, however, as illustrated by the fact that in one case an erythema rapidly developed whenever the patient was spoken to at all brusquely. He states that the eruptions are not of any gravity, as a rule, the only matter of importance being that they are not infrequently associated with albuminuria.

P. Remlinger[2] believes that the **desquamation** occurring in typhoid fever is a trophic disorder of the skin which is comparable to the loss of hair seen in that disease.

Schaefer[3] gives a general consideration of the question of **typhoid ulceration of the pillars of the pharynx.** It is recognized that this occurs with considerable frequency. Schaefer considers that it is the result of the dryness of the mouth, the relative immobility of the pillars, and the compression exerted upon them; the latter, acting upon tissues which readily undergo necrosis, owing to the infection present, causes ulceration of the pillars. It is perhaps to some extent also an actual typhoid disease of the lymphoid tissues. His examination of 5 cases, however, showed in each instance the absence from the local lesions of the typhoid bacillus. He has found these ulcerations present in 1 out of every 5 or 6 cases of typhoid fever. They are much more common in men; possibly as a result of the use of tobacco. Their appearance has no relation to the severity of the disease. The ulcers have clear-cut edges, and are very different from tuberculous ulcerations, mucous patches, chancres, or aphthæ, and their presence has been used at times to confirm a diagnosis of typhoid fever.

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[1] Am. Jour. Med. Sci., July, 1899.

[2] Rev. de méd., May 10, 1900.

[3] Thèse de Paris, 1899.

**Complications.**—Potain[1] discusses the occurrence of typhoid fever **in direct succession to influenza,** and reports 6 cases of this character. In all these there was an onset characteristic of influenza, and the cases ran the course of influenza for a variable period. Then doubtful signs of typhoid fever developed, and the typical course of typhoid fever ensued, with positive serum reaction. He believes that there was no doubt of the existence of influenzal infection in these cases, basing this view most strongly upon the fact that the rose spots appeared only after from 11 to 31 days had elapsed. The cases were not unusually severe; in fact, they ran a mild course. In discussion Le Gendre described 2 similar cases in which there was an influenzal onset and afterward a distinct course of typhoid fever. Both these persons had been clearly subjected to infection with typhoid, one of them, a physician, in studying the pathologic anatomy of typhoid fever, and the other in nursing a typhoid case. He believes that it was evident in these cases that the influenzal infection made the subjects more susceptible to infection with typhoid fever. Several others in discussion mentioned their experiences with typhoid following influenza. [We have repeatedly seen this in the past year, and have become convinced that an **influenzal infection increases the susceptibility** to typhoid fever.]

A. McPhedran[2] notes that **diagnosis of perforation** in typhoid fever is sometimes extremely difficult. He describes 2 interesting cases. The first patient had been treated for syphilis, and was taking potassium iodid. He had some indisposition which was attributed to disturbance of digestion by the iodid. He had no fever and his pulse was normal, but later he had pain in the abdomen, and there was evidently some fluid in the peritoneal cavity. Death occurred, and necropsy showed 2 small typhoid ulcers in the ileum, one of which had perforated. This was a strikingly mild case of apyrexial typhoid without any evident symptoms of perforation. In the second case the man was evidently in the midst of an attack of typhoid, and he had pain without any definite signs of perforation; but since there was persistent tenderness of the abdomen, he was operated upon, and a perforation was found, almost completely walled off. The man died. **Persistent pain** is considered the **most constant symptom** of perforation.

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[1] Gaz. des Hôp., June 1, 1900.

[2] Phila. Med. Jour., Mar. 3, 1900.

G. B. Shattuck, J. C. Warren, and F. Cobb [1] report **24 cases of typhoid fever** in which **laparotomy** was undertaken because of the occurrence of peritonitis. From their study of these cases they decide that oftentimes infection of the peritoneum can not be diagnosed until it is so wide-spread as to be inevitably fatal. In some cases of mild typhoid fever sudden perforation may occur, and the process may be at first localized. In such cases immediate operation gives good chances of a successful outcome, but general infection is likely to occur even within the first few hours, and there is practically no chance of a spontaneous limitation of the process by adhesions. In mild cases the evidences of perforation are usually obscure, and consist almost entirely in local pain, tenderness, and spasm, and in the occurrence of leukocytosis. The presence of these symptoms should lead to operation, or at any rate to consideration of operation. Pain is an important sign of beginning infection of the peritoneum, and the authors consider the occurrence of leukocytosis an exceedingly valuable sign. In order to make this of practical importance, however, the leukocytes should be very frequently counted in suspected cases. Of the 24 cases of peritonitis reported, 7 were not due to perforation. In 14 cases the symptoms were of gradual onset, while in only 7 were they sudden. In 6 cases recovery ensued, but in 3 of these no infection of the peritoneum was found at the operation.

Beschorner,[2] in discussing the **relapses in typhoid fever** during the last 17 years in the Dresden City Hospital, notes that they occur chiefly in young persons, but that when they occur in older persons the prognosis is much worse. The mortality in general was 7.38 % ; the mortality in relapses, 2.6 %. The relapses were more serious when they began after only one fever-free day. It was found that if the leukocytes persisted at a subnormal point in the fever-free period, relapse might soon be expected, and persistence of the enlargement of the spleen was also a sign of importance in this connection, though it did not always indicate a relapse.

P. Horton-Smith,[3] in his lectures on the typhoid bacillus, states that it is fairly well shown that relapses are due to the fact that the patient has acquired sufficient immunity to destroy most of the bacilli, but some remain, and absorption of toxins from the alimentary canal takes place, this leading to reinvasion of the organism and reinfection. He insists that this emphasizes the **necessity for withholding solid food** until some time after the temperature has become normal. Autointoxication is also probably of importance in giving opportunity for the primary attack. He then reviews the evidence that the typhoid bacillus may cause a general disease without intestinal lesions, and also the evidence of typhoid fever in the fetus, and then discusses local infection in typhoid fever, concluding that there is often sufficient evidence that the disease is a local one. Concerning the complications observed at St. Bartholomew's Hospital in the last 30 years he notes that there is but 1 case of meningitis reported. Ulceration of the larynx occurred in 25 % of cases, probably as a result of infection by other organisms. Lobar pneumonia occurred in 5 %. He believes that there is no satisfactory evidence as yet that lobar pneumonia is ever purely typhoidal. Endocarditis was seen but 4 times. It is usually, but not always, due to secondary infection. He reports 1 case that began with a violent acute nephritis and typhoid bacilluria, and then ran the usual course of typhoid fever. He has seen 17 cases of **bacilluria** in 45 cases examined; excluding the selected cases, the figures would be 11 out of 39, and he believes that bacilluria occurs in about one-fourth of all cases of typhoid fever. This condition may often be recognized with the naked eye by observing that the urine is turbid, and has a peculiar shimmer like that of a broth culture. It rarely occurs before the third week, and may occur very much later. Pus is often found in the urine, but albumin is usually absent. He considers that this condition can not be due to filtration from the blood or to local suppuration, as a rule, but that it is due to the multiplication of stray bacilli which gain entrance into the urine. It is usually cured by local treatment. He believes that bacilluria as a result of infections other than typhoid is a much more common condition than is usually thought. If the bladder is injured in any way, cystitis is likely to result. The typhoid bacillus is often found in pure culture in bone abscesses, but only infrequently in suppurations elsewhere. That a patient may give rise to infection long after the disease is past is shown by the fact that the typhoid bacilli have been found in pus as long as 14½ years after the occurrence of the disease.

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[1] Boston M. and S. Jour., June 28, 1900.

[2] Festschr. z. Feier d. 50-jähr Best. d. Krankenhaus Dresden-Friedrichstadt.

[3] Lancet, Mar. 31, 1900.

N. B. Gwyn,[1] in discussing examination of the urine for typhoid bacilli, divides cases of **typhoid bacteriuria** according to the local signs. Some cases show nothing abnormal with the exception of the presence of the bacteria. Most cases show some signs of irritation of the urinary tract, while in others these symptoms are prominent. Gwyn's experience comprises 10 cases, in 3 of which bacilli were obtained and identified before the Widal reaction became positive. They were therefore of diagnostic importance. The urine was always acid and usually turbid, and upon examination showed myriads of bacteria. In 1 case of typhoid septicemia with a duration of 10 weeks the examination of the urine led to the diagnosis of typhoid infection. He does not consider that the examination of urine will ever be a very important diagnostic aid, however, as the bacilli are found in only from 20 % to 30 % of cases. He considers that irrigation with bichlorid 1 : 50,000 is the best treatment. Urotropin is sometimes useful, but in some cases he has found it ineffectual. He believes that a microscopic examination of the urine should be made in all cases of typhoid, as one can usually recognize a marked bacteriuria in this way. For safety, the urine should be disinfected in all cases.

T. R. Brown [2] reports a case in which celiotomy had been done, and in which there had probably been slight injury of the bladder during the operation. It was necessary to catheterize the patient, and **cystitis** developed. Bacteriologic examination of the urine showed the presence of the typhoid bacillus. The patient had had typhoid fever 35 years before, so that it seemed impossible that it had been an auto-infection. Brown believed that the infection occurred from the catheter, and that the bladder was especially susceptible because of the preceding trauma.

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[1] Phila. Med. Jour., Mar. 3, 1900.

[2] Med. Rec., Mar. 10, 1900.

R. B. H. Gradwohl[1] reports a case which ended fatally, with great enlargement of the liver, tenderness over this organ, and severe dyspnea. The autopsy showed a large **abscess** involving about one-half **the liver.** The bacteriologic examination showed the presence of a bacillus with the characteristics of the typhoid bacillus, which reacted to serum from a case of typhoid fever. The man had had an attack of typhoid fever 6 years previously.

Wallgren[2] describes the case of a woman of 39 who had had typhoid fever 3 years before her entrance to the hospital. She presented a mass in the abdomen which had been present for 5 years and had progressively increased in size. This proved to be an ovarian cyst, and celiotomy disclosed the fact that the contents were purulent. Bacteriologic examination showed the presence of a pure culture of the typhoid bacillus. It was evidently a **typhoid infection of the ovarian cyst.**

J. M. Da Costa[3] describes 3 cases of **typhoid cholecystitis** which ended in recovery. The first case had numerous other complications, but the cholecystitis was extremely mild and the diagnosis seems somewhat questionable. In the second case there was a mild but definite cholecystitis; in the third, a very severe one. All recovered, however, without operation. [In several cases in our own experience post-typhoidal cholecystitis has been a protracted disease, though mild in its manifestations.]

H. R. Brown[4] describes a case of typhoid fever in which the disease came on with severe symptoms of **acute nephritis.** Typhoid fever afterward became evident. He considers the case rare. [A reference to recent literature will show, however, that it is not very rare to see cases of typhoid fever which often begin with such pronounced nephritic symptoms as to cause them to be mistaken for acute nephritis. The typhoidal nature of the attack is often overlooked.]

B. F. Stahl[5] describes 10 cases of **gangrenous dermatitis** which occurred in a series of 144 cases of typhoid fever in soldiers treated in St. Agnes' Hospital, Philadelphia. It is a strikingly large number when it is considered that this complication is ordinarily found only rarely. Gangrene developed when the most severe period of the disease had passed; it was associated with grave anemia. The first heart-sound in 5 of the cases was extremely feeble, and mitral regurgitation was present in 2 cases. Cyanosis in one instance, and in another hyperemia, preceded the gangrene. Arteriosclerosis was present in decided degree in most of the cases. The area of gangrene was usually of oval shape, and at postmortem on 2 fatal cases there were found ecchymotic areas in the stomach and infiltration of the lungs and of one kidney; in another instance both kidneys and the spleen were found infarcted. All these facts point to embolism or thrombosis as the cause of the condition. Bacteriologic investigation showed the presence of staphylococcus aureus and staphylococcus albus. The lesions, with one exception, did not involve the extremities—a fact in contrast with previous reports. The trunk was most commonly involved; 3 of the 10 cases were fatal.

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[1] Interstate Med. Jour., Nov., 1899.

[2] Arch. f. Gynaek., 1899, vol. LI, p. 15.

[3] Am. Jour. Med. Sci., Aug., 1899.

[4] Brit. Med. Jour., Jan. 27, 1900.

[5] Am. Jour. Med. Sci., Mar., 1900.

A. A. Hubbell,[1] in discussing **eye complications** in typhoid fever, mentions inflammations of the conjunctiva and iris, suppuration in the deeper eye structures, opacity of the vitreus or lens, infections of various parts of the eyeball and orbit, hemorrhage, thrombosis, and hemianopsia. As sequels one may see ocular palsy and atrophy of the optic nerve. The cause may be direct infection, but is usually a toxemia. The conjunctiva often becomes inflamed as a result of the lessened lacrimal secretion and of the profound exhaustion, which causes the patient to lie with his eyes open without winking. The eye should be carefully attended to during the course of the disease. The prognosis of eye affections in typhoid fever is more grave than when they occur in health.

H. Salomon [2] made a series of **examinations of the optic discs** in typhoid fever, and found that in the early stages of the disease there was often an appearance similar to that seen in cases of increased intracerebral pressure, the disc being somewhat injected, the veins dilated and tortuous, and the borders indistinct. Lumbar puncture, which was carried out in some of these cases, showed a pressure ranging from 180 mm. to 250 mm. of water. The fluid was sterile whenever examined, and showed no agglutinating power. Salomon thinks that cerebral symptoms in typhoid in the early stages are usually due to an excess of cerebrospinal fluid. He thinks that this explains the slow pulse of the early stage of the disease.

A. G. R. Foulerton and H. C. Thomson,[3] in discussing the **causation of nervous symptoms** in typhoid fever, state that they may be conceived to be due to action of the typhoid bacillus or to secondary infection, and it is difficult clinically to determine which is the cause. If they are due to the typhoid bacillus, it may be through direct infection of the central nervous system, causing typhoid meningitis, or to toxemia. They discuss the question as to the existence of alterations in the ganglion cells in local typhoid infection or in typhoid toxemia. The experimental inoculation of rabbits with living cultures of typhoid bacilli or the injection of typhoid toxins caused slight alterations in the ganglion cells when hardened with formalin and stained in methylene-blue, but the alterations were not seen in most of the cells, and were slight and of irregular character. The same was true in a case of typhoid toxemia with cerebral symptoms and in one of typhoid meningitis, both of which ended fatally. In no other case were any changes found in the ganglion cells with the exception of a few slight and inconstant alterations. The case of meningitis was of some interest because of its rarity. It occurred in a boy of 12, who was admitted in an almost unconscious condition, and who paid little attention to surroundings or to questions. He had a frowning expression, his eyes were rolled about a great deal, and the diagnosis at first was thought to be tuberculous meningitis, but the Widal reaction was subsequently obtained. Later he had retraction of the head and facial paralysis. When he died, the meninges were found much congested, and cultures from these membranes showed the presence of a bacillus having the characteristics of the typhoid bacillus and reacting with blood of a rabbit immunized against typhoid infection in a dilution as high as 1 : 1000.

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[1] Med. News, Nov. 11, 1899.

[2] Berl. klin. Woch., Feb. 5, 1900.

[3] Lancet, April 21, 1900.

A. Schiff[1] reports a case of typhoid fever in a boy of 19 in which, after passing through 5 days of entirely normal course, the patient was observed to be much worse, and it was discovered that he had complete paralysis of sensation of all qualities from the level of the third costal cartilage downward. Also he had entire motor paralysis of the legs and almost complete paralysis of the arms. These symptoms were followed by death within 18 hours, a diagnosis of transverse myelitis at the level of the fourth or fifth cervical segment having been made. The postmortem disclosed an **acute hemorrhagic myelitis,** the changes consisting of scattered small hemorrhages, a hemorrhagic infarct in the fourth cervical segment which had destroyed practically the whole of the gray matter of the cord, enormous dilation of the vessels from the fifth to the eighth cervical segment, and degeneration of the ganglion cells in the cervical cord even when there was no hemorrhage in the immediate neighborhood. Micro-organisms were not found in any part of the cord. The hemorrhage was attributed to toxemia. Five other cases of acute infections of the spinal cord in association with typhoid fever seem to be the only ones so far reported. In 4 in which autopsy was carried out the results were as follows: In one there were no macroscopic changes; in another typhoid bacilli were found in the cord without any macroscopic or microscopic changes; in the third there were capillary hemorrhages and areas of softening; in the fourth there were hemorrhagic areas in the medulla.

H. C. Thomson[2] describes a case of **acute glossitis** which complicated typhoid fever at about the end of the fourth week. The swelling increased rapidly and caused death.

H. M. Fisher[3] describes a case of typhoid fever in which the signs of **meningitis** came on. The patient died, and the autopsy disclosed a general meningitis. In the exudate a bacillus corresponding to the typhoid bacillus was found.

T. A. Claytor[4] describes 2 cases of typhoid fever in which general and repeated **convulsions** occurred. There were no evidences of meningitis or encephalitis in either of the cases, and neither of the patients had any signs of epilepsy. There was also no evidence of uremia, with the possible exception that in one case the urea was at one time much reduced on the day of the convulsions and that a small amount of urine was passed; but no albumin or casts were present. The convulsions were thought to be due to severe toxemia.

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[1] Arch. f. klin. Med., Bd. LXVII, Hefte 1, 2.

[2] Lancet, June 23, 1900.

[3] Phila. Med. Jour., Mar. 3, 1900.

[4] Phila. Med. Jour., Mar. 31, 1900.

F. Mühling [1] also describes a case of typhoid fever in which **epileptiform convulsions** appeared during convalescence.

E. G. Janeway,[2] in reporting unusual complications in typhoid fever, mentions 3 cases of **tetany,** a number of instances of **psychosis,** and some instances of **suicidal tendency.** Of eruptions he noticed that herpes labialis and a petechial rash occurred in one case, which was also complicated by jaundice.

J. M. Da Costa [3] describes a case of **aphasia** during the course of typhoid fever in which the disturbance of speech was not constant. The cause was probably hysteria.

R. W. Lovett and C. F. Whittington [4] discuss the **typhoid spine** —a condition which follows typhoid fever and somewhat resembles Pott's disease. The chief symptoms are marked pain and tenderness of the spinal column. There is some question as to whether it is a pure neurosis or a spondylitis. They describe a case, in which typhoid fever had probably occurred, in which there was marked pain and tenderness of the spinal column with increased knee-jerks, slight ankle-clonus, and some patches of anesthesia on the thigh. Hysteria was probably an important element in the case, though it seemed probable that there was some pressure on the cord. The patient, however, recovered entirely. [In one case under our observation similar symptoms, persisting for some time during convalescence, were entirely relieved by free purgation.]

Könitzer [5] describes a case of diffuse **spondylitis** in a man of 25 which occurred after recovery from typhoid fever. He had some preliminary pain, and there was a sudden onset of severe pain in the loins, which radiated backward and down the thighs. The patellar reflexes were at first exaggerated and then lost, subsequently returning to normal. The patient had some fever. There was no swelling in the back. The patient soon grew better and recovered, but had pain on stooping for some time.

J. F. Schamberg [6] reports the occurrence of **typhoid fever in a leper.** He has found no other instance of typhoid fever in lepers. Calmette's antivenomous serum was used in the treatment of the leprosy, but without result.

F. A. G. Murray [7] reports a case of typhoid fever complicated by **suppuration of the thyroid gland and by orchitis.** The patient was a man of 22, a bugler, who had been at Camp Meade. The thyroid gland was swollen, but the swelling subsided about 3 weeks later. The gland became swollen again and very tender, and the man had marked leukocytosis. Incision was made and pus was evacuated. The bacteriologic examination showed a pure culture of a bacillus having the characteristics of the typhoid bacillus, though the reaction of these bacilli with the patient's blood-serum was not satisfactory. Later the man had a marked orchitis.

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[1] Münch. med. Woch., Feb. 13, 1900.
[2] Med. News, Dec. 9, 1899.
[3] New Orl. M. and S. Jour., Dec., 1899.
[4] Boston M. and S. Jour., Mar. 20, 1900.
[5] Münch. med. Woch., Aug. 29, 1899.
[6] Jour. Am. Med. Assoc., Jan. 20, 1900.
[7] Phila. Med. Jour., Dec. 16, 1899.

D. J. M. Miller [1] reports a case of **swelling of the submaxillary glands** in the course of typhoid fever without any swelling of the parotid gland. Isolated swelling of the submaxillary glands in this disease is rare, but in this case it was extremely marked.

Etienne [2] describes a case of typhoid fever in which on the fourth day of the disease there was a **peculiar eruption** of flat reddish spots which were very different from the usual rose spots of typhoid fever, and made him think of the possibility of typhus fever. The serum reaction was positive later. Examination of the blood showed the presence of large numbers of a bacillus which formed small transparent colonies and was not pathogenic. The patient finally died in coma, a skin eruption of purpuric spots having appeared before death; there were also severe hemorrhages from the intestines. Typhoid fever was found to be present and the condition was attributed to coincident infection with the bacillus previously mentioned. This case demonstrates the gravity of **associated affections** in typhoid fever even when the organism causing the infection is not pathogenic. [It must, however, be recalled that simple typhoid infection occasionally causes hemorrhagic manifestations. The pathogenicity of the bacillus found in Etienne's case was unproved and somewhat doubtful.]

A. A. Eshner [3] reports a case in which the patient, a woman of 28, had tuberculosis of the lungs and developed typhoid fever, and the latter was complicated by pneumococcus pneumonia, which occurred in the later stages of the typhoid fever. The case thus constituted an instance of **triple infection.**

J. L. Bevans [4] reports a case of **mixed typhoid and malarial fevers** in a soldier who had served in Cuba, and had apparently had slight malaria there. In October, 1899, he went to bed with symptoms of typhoid fever, and characteristic spots developed on the seventh day. There were chills for several days. At this time the ring form of the quotidian parasite was discovered. The subsequent course of the fever was thought to be typhoid, and the Widal reaction was positive on November 9th. The chills in the early part of the attack were controlled by quinin, and after the use of this drug the malarial organisms disappeared.

D. Harris [5] describes the case of a sergeant who was taken ill on February 22d with chill and malaise. Owing to the prevalence of influenza he was isolated. The next day he showed the physical signs of pneumonia. Later he had a diphtheric membrane over the anterior

[1] Univ. Med. Mag., July, 1899.

[2] V Congres Français de Méd. interne, 1899.

[3] Am. Jour. Med. Sci., July, 1899.

[4] N. Y. Med. Jour., Feb. 10, 1900.

[5] Lancet, April 7, 1900.

pillars of the fauces. A few days after this his appearance was that of typhoid fever; some rose spots and enlargement of the spleen were made out, and there was blood in the stools. His temperature reached normal 2 weeks after he was taken sick. The case is reported as one of **concurrent pneumonia, diphtheria, and typhoid fever.** [The presence or absence of the Widal reaction and diphtheria bacilli are not reported upon, and there is no proof that the three diseases were present, but it seems probable from the account that it may have been a case of pneumonotyphoid.]

**Diagnosis.**—W. Osler [1] gives a general discussion on the diagnosis of typhoid fever, particularly of the more obscure forms of the disease. Variations in intensity of infection produce changes in the course of the disease from a mild so-called simple continued fever to an acute violent typhoid septicemia. The Widal reaction is the most important aid in the study of cases of extreme mildness or of extreme severity. Among the most obscure cases are those in which there is an early and pronounced localization of the infection elsewhere than in the intestine,—for instance, in the pleura or lung, and even more particularly in the kidney,—with the production of a typhoid nephritis and with perhaps the absence of enteric symptoms for 10 days or more. These are very prone to be incorrectly diagnosed. Chills are particularly likely to lead to errors in diagnosis, as many men are in the habit of interpreting them at once as an evidence of malaria. Actual mixed infection is extremely exceptional; and as to the **diagnosis between typhoid fever and malaria,** Osler states that above Mason and Dixon's Line a practitioner may usually conclude that any intermittent fever which resists quinin is not malarial, and that any continued fever is not malarial, because the estivoautumnal form of malaria is rare in this region. In the regions where estivoautumnal fever may occur it is differentiated from typhoid by remembering that it has at all times marked remissions, while typhoid is very regular, and in malaria there is a pronounced early anemia with a sallow complexion. Of course, when they are obtained, the Widal reaction and the discovery of plasmodia are extremely valuable.

E. Stuver [2] believes that **mountain fever** is mere **atypical typhoid fever.** The temperature is usually fairly characteristic, though sometimes irregular. An eruption is usually present. There are commonly marked pains in the muscles and back. Intestinal hemorrhage occurs only rarely. He treats the condition by the use of calomel and intestinal antiseptics, with a limited use of cold baths and colonic injections of iced water.

M. W. Richardson [3] reports 6 cases of typhoid fever in which he used the method of Neufeld in **investigating the typhoid spots for bacilli.** After rubbing the skin with alcohol and ether he froze the spot with ethyl chlorid in order that the incision might be painless, and also that the blood might be driven out of this section, thus making the probabilities of success greater. In 5 out of 6 cases bacilli were obtained, and the failure is attributed to the fact that only 2 spots were incised and no second cultivation was attempted. In 2 cases success was attained only upon the second cultivation. Richardson recommends that at least 5 or 6 spots should be incised whenever this investigation is carried out. On the average, positive results were secured by this method about 6 days sooner than by the Widal method.

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[1] N. Y. Med. Jour., Nov. 4, 1899.

[2] Med. News, Nov. 4, 1899.

[3] Phila. Med. Jour., Mar. 3, 1900.

Curschmann,[1] in examining the typhoid spots for bacilli, found them in 14 out of 20 cases. This investigation may prove of value in the diagnosis of typhoid, and is not a difficult procedure.

J. A. Care [2] describes the **method of Piorkowski** for distinguishing the typhoid bacilli by growing them on a medium composed of 100 parts of urine which has undergone ammoniacal fermentation and to which 0.5 part of peptone and 3.3 parts of gelatin have been added. The subsequently sterilized culture-medium is placed in tubes and the tubes are inoculated from the feces of the patients to be studied. The contents of the tubes are then poured on Petri dishes. The dishes are kept at a temperature of 22° C. The colonies are said to be characteristic in 24 hours, and appear as transparent filamentous bodies, contrasting with the characteristic closely formed colonies of colon bacilli. The characteristic colonies are said to be composed of typhoid bacilli alone, and have been observed as early as the third day of indisposition. Typhoid bacilli have also been grown upon the media when inoculated from the spleen of a patient dead of typhoid. The method is believed by Piorkowski and his associates to be distinctive of typhoid fever.

Schütze [3] discusses the diagnostic value of this method of demonstrating typhoid bacilli in the feces. Four cases of typhoid fever in which the diagnosis was somewhat difficult are here recorded in which Piorkowski's method gave a positive result. In 3 cases the diagnosis was confirmed by necropsy and in the fourth case by the course of the disease; in a fifth case typhoid had been thought probable, but the result of Piorkowski's method had been negative and the case proved to be one of pelvic cellulitis. Schütze has made studies of the colonies which develop upon Piorkowski's medium and has found that they are actually typhoid bacilli.

E. Unger and E. Portner [4] found that Piorkowski's medium is not a satisfactory method of making a diagnosis of typhoid fever by cultures. The growths of the typhoid bacillus are, however, usually distinctly different from those of the colon bacillus; and if cultures with long filamentous processes do not appear, this speaks against typhoid fever, and if many of these peculiar cultures are present, it is in favor of typhoid fever.

J. R. Arneill [5] reports the results of the use of the **diazo reaction** in Dock's clinic in the past 6 years. Examinations in 405 cases are reported, of which 81 were positive. Of 19 cases of typhoid fever, it was negative in 2 which came in late, and in one other which was a mild attack; in all the others it was positive. It is considered that the reappearance of the diazo reaction points to a relapse; this is of some prognostic importance, since the reaction usually appears the latter part of the primary attack. The duration of the reaction is thought to be some indication of the probable length of the fever; when it tends to disappear, the fever is likely to decline early. Of 82 cases of tuberculosis, 42 showed the reaction. With Michaelis, Arneill considers that the presence of the reaction in pulmonary tuberculosis is a bad sign. It is thought to be of importance in diagnosis between cirrhosis of the liver and tuberculosis of the peritoneum, since when positive, it points to tuberculosis. He believes that there is convincing evidence of the value of the reaction in the diagnosis and prognosis of typhoid fever, and in the prognosis of anemia, diphtheria, and septicemia, and particularly of tuberculosis.

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[1] Münch. med. Woch., Nov. 28, 1899.
[2] Phila. Med. Jour., Oct. 14, 1899.
[3] Zeit. f. klin. Med., Bd. XXXVIII, Hefte 1, 2, and 3.
[4] Münch. med. Woch., Dec. 19, 1899.
[5] Am. Jour. Med. Sci., Mar., 1900.

Guillemin,[1] in reporting upon a study of the diazo reaction, stated that the parallelism between the temperature curve and the diazo reaction, which was considered by some authors to exist, was in his experience very rare, and even then not constant. But the diazo reaction does appear to be an index of the general condition of the patient, and hence is probably an **index of the degree of elimination of toxins.** If the kidney is in bad condition, the toxins are not well eliminated and the diazo reaction diminishes. In typhoid fever he considers that it indicates well the condition of the patient and the possibility of a relapse. In a case of puerperal fever the diazo reaction followed the course of the fever, and thus indicated the amount of the intoxication.

Karcher [2] reports that in the epidemic of typhoid fever which occurred in Basel in 1898 only 32.6% of the cases showed the diazo reaction, and the **reaction was found to be of little worth,** both for this reason and because it was seen in a number of other diseases which appeared during this epidemic. The Widal reaction, on the other hand, was positive in almost all the cases.

P. Horton-Smith, [3] in discussing the typhoid bacillus and typhoid fever, notes under the subject of the **serum test** that he has with others found marked variations in the clumping power of different typhoid bacilli. For instance, one bacillus obtained in pure culture from the spleen of a fatal case of typhoid did not clump in a typhoid serum diluted to 1 : 20. Marked differences were found in other bacilli. This probably, however, does not indicate that there are different varieties of bacilli. So far as they have been carried out, Horton-Smith's experiments indicate that there are but slight differences in agar cultures. His work agrees with that of Funck in showing that the toxin of the typhoid bacillus is contained in the body of the bacillus. Filtrates of cultures were innocuous. Concerning the distribution of the bacillus, he notes that he has found it in the bile in 9 of 10 cases, in the bone-marrow in 4 of 6 cases, only twice in the kidneys out of 8 cases, and only once in the lungs in 5 cases. Of 12 cases in which the blood was examined postmortem, he found the bacillus in 10, one of these being a case that had ended on the fourteenth day from the general severity of the disease and heart failure—evidently a profound septicemia. In his concluding lecture [1] he considers the Widal reaction. He decides that the danger of confusion with an infection by the Gärtner bacillus has been overestimated and is chiefly theoretic. The serum of a guinea-pig which had been immunized against Gärtner's bacillus reacted in a dilution of 1 : 2000 against this bacillus; but with the typhoid bacillus practically no reaction was obtained in a dilution of 1 : 20. In ordinary work Horton-Smith recommends a 1 : 20 dilution with a 1-hour limit. If the result be doubtful in any one case, it is well to use a **dilution of 1 : 100**; but high dilutions should not be used in ordinary work, as many cases that are really typhoid will not react in a high dilution. His experience with 546 tests is recorded. Over 200 were made in cases that proved to be non-typhoidal, and in only one of these was the reaction obtained, and in this case it was shown afterward that typhoid fever had occurred 4 months before the disease for which the patient was admitted to the hospital. There are certain cases, perhaps 3 %, in which the reaction never appears, and he describes 2 such cases. In the first, death occurred on the seventeenth day, so that perhaps the reaction would have appeared later had the boy lived. In the second case the bacilli were found in the stools, and all the appearances were those of typhoid fever. Recovery occurred, and the reaction was never found. The duration of the reaction was from the time of the appearance of defervescence to over 2 years; usually it disappeared within a year. Horton-Smith suggests that the long persistence of the reaction in some cases may be connected with the continuance of the bacilli in the body. He considers the reaction **worthless in prognosis.** He believes that it is shown that the agglutinating substance is probably a ferment, and that this is probably produced by the cells of the body, particularly by the spleen, the lymph-glands, and the bone-marrow under the stimulus of the infection. The reaction is undoubtedly a reaction of infection, but is probably connected with immunity. Later in the disease bacteriolytic properties appear in the blood, and finally the further growth of the bacilli is checked and they are destroyed. He considers the prospects of success from Wright's method of preventive inoculation good. He records 10 cases in which urotropin was used to overcome typhoidal bacilluria with success. He describes the immediate effects of this drug as astonishing, and presents interesting illustrations of cultures before its use and attempted cultures afterward, the latter usually being negative. He believes that the drug should be regularly used even though good nursing may be secured, in order to prevent the spread of infection, and particularly insists upon its value in the management of cases under bad hygienic surroundings in the poorer classes.

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[1] Compt. rend. de la Soc. de Biol., Jan. 17, 1900.

[2] Cor.-Bl. f. schweiz. Aerzte, Sept. 1, 1899.

[3] Lancet, Mar. 24, 1900.

A. O. J. Kelly and A. A. Uhle [2] describe the results obtained in

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[1] Lancet, April 14, 1900.

[2] Phila. Med. Jour., Mar. 3, 1900.

**1080 Widal reactions.** In 411 cases of typhoid in which the test was performed once, only 360 gave positive results. Of 110 cases in which the test was performed twice, 55 were positive twice; 10 negative twice; 38 negative once, then positive. Of 27 cases in which the test was performed 3 times, 10 were always positive, 9 positive twice, 3 positive only upon the third test. There is a series of diseases reported in which positive reaction was obtained though the patients were not suffering from typhoid; some of them, however, had had typhoid previously. Kelly, in commenting upon these results, insists upon the importance of knowing the dilutions in interpreting the results of the Widal tests. Hence he considers the dried blood method uncertain, though agreeing that it is the only one that can be used by public health-officers and in similar work. He considers that the test is not pathognomonic unless it is obtained with high dilution; but if high dilutions can be accurately obtained, it is the best test we have, and must be considered of very great diagnostic import.

J. J. Curry,[1] in reporting on the blood examination for typhoid fever and malarial fever in the army hospitals at Fort Meyer, Va., and Savannah, Ga., states that **95% of cases** which were clinically diagnosed as typhoid fever **gave the Widal reaction,** and practically all cases not typhoid fever gave no reaction. The test was considered wholly satisfactory, though in about 50% of cases no reaction was obtained until about the end of the second week. There were a few cases of mixed typhoid and malarial infection, and in several instances malarial parasites were found during the convalescence from typhoid fever.

A. R. Guerard [2] considers the Widal reaction satisfactory in a dilution of 1 : 10, made approximately by observing the color of diluted dried blood. He prefers, however, the **serum from a fly-blister;** and if this reacts within 5 minutes in a dilution of 1 : 10, he considers the reaction absolutely positive; if it does not react in this time, it is probably negative. With proper care in performing the test he considers it extremely reliable, and thinks that if properly done it is never obtained in diseases other than typhoid fever.

R. T. Hewlett and S. Rowland [3] report **a new method** for the accurate **quantitative performance of the Widal reaction.** They take ordinary vaccine tubes, carefully sterilize and dry them, and then fill them one-half to two-thirds full with blood, sealing them by heat. When the test is carried out, the length of the blood column is measured; the end of the tube is then filed off and its diameter measured by the microscope and micrometer scale. In doing this, the tube is placed between 2 iris diaphragms, which close upon it—one of them placed on the stage and the other on the substage. The closure of the diaphragm leaves only a narrow rim of light, corresponding to the glass of the tube, and the rest is in darkness. The width of this rim of light is measured, and the amount of blood contained in the tube is then estimated by multiplying the square of the diameter by the length of the column, and this by the figure 0.7854. The tube is then placed in a thick-walled glass tube of larger lumen, the lower end of which is submerged in the correct amount of diluting medium. The capillary tube is then crushed by a soft iron plunger passed through the outer tube, the latter is washed in the diluting medium, and the test is carried out.

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[1] Boston M. and S. Jour., Nov. 23, 1899.

[2] N. Y. Med. Jour., April 21, 1900.

[3] Brit. Med. Jour., April 28, 1900.

Sabrazès and Brengues [1] have investigated the **possibility of drugs having an influence upon the agglutinating action** of the blood-serum upon the typhoid bacilli. So far as their studies have gone, however, the results have been negative.

P. Courmant and Cade [2] describe an instance in which typhoid fever occurred in a nurse, and the **infant's blood-serum agglutinated** the typhoid bacillus. They consider that there was no origin for this other than **through the milk** of the nurse. The child's blood agglutinated in a dilution of only 1 : 10, the blood of the nurse agglutinated at 1 : 200, and the milk agglutinated in a dilution of 1 : 30.

Etiénne [3] considers that in the course of typhoid fever in the mother the fetus may be infected, may remain entirely unaffected, or it may receive typhoid toxins and react against these. The possibility of the last condition is demonstrated by a case in which he observed a higher agglutinating power in the blood of the fetus than in that of the mother; hence he considered that the agglutinating substance had not passed from the mother to the fetus, but that the child had reacted to produce the agglutin. No bacilli were found in the tissues of the child, hence the reaction of the child's tissues must have been produced by the toxin. The agglutinating power was the same in the blood of the fetus as in the amniotic fluid, which tends to show that the amniotic fluid is of fetal origin.

Paris [4] reported an **epidemic of typhoid fever** in women at the time of the termination of **pregnancy.** In some of the patients labor was passed through 2 or 3 days before any manifestation of the disease appeared in the mother, and yet the infants presented the same symptoms as those seen in the infants whose mothers were in the course of typhoid fever at the time the children were born. It seemed probable that an amount of toxin which is incapable of having any apparent influence upon the adult tissue was present at the time of the birth of the children, and was sufficient to cause disturbance in the more delicate organism of the fetus.

**Treatment.**—Vaquez, [5] in discussing the **diet** of typhoid cases, recommended the use of 3 egg-yolks, and 1 or 2 small teaspoonfuls of somatose, 2 small glasses of wine-jelly or beef-juice, 1 plate of farinaceous soup, some bouillon, and 2 liters of milk as a day's diet, the meat-juice being rapidly increased when the temperature begins to fall. In discussion Siredey, while accepting the value of freer feeding in some cases of typhoid, insisted that it was often possible to send the temperature up by giving a single yolk of an egg. Merklen believed that the diet proposed could not be used in the majority of cases. He insisted that milk is the proper diet in most cases, and that if this is not well borne, one should give bouillon instead, or a mixture of milk and bouillon. He was convinced that he had seen evidences of autointoxication and definite recrudescence of fever as a result of disturbance of digestion, and that he had even seen relapses follow such disturbance. Widal believed that milk was the aliment of choice in typhoid fever, and considered that the patients usually had not sufficient gastro-intestinal secretion to admit of their taking of much freer diet. A point of value in the use of milk is that it causes free diuresis. [In our experience the only form of diet not likely to cause elevation of temperature is milk. Broths, eggs in milk, and even albumen-water, may cause a rise of temperature after the fever has practically subsided.]

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[1] Compt. rend. de la Soc. de Biol., Nov. 25, 1899.

[2] Ibid., July 9, 1899.

[3] Ibid., Nov. 4, 1899.

[4] Ibid., Jan. 20 and 27, 1900.

[5] Gaz. des Hôp., Feb. 16, 1900.

R. H. Fitz[1] analyzes the record in cases of typhoid fever which had been admitted to the Massachusetts General Hospital in the past 78 years. The mortality during this time has been about uniform, ranging from 13% to 16%, as a rule. The complications also have not varied greatly in this time. Up to 1839 the treatment was active medication, with free use of drastic purges and a liberal diet. After this time the treatment was more gentle and the diet was reduced. Fitz does not think that there is any indication from his study that the mortality of the disease has been greatly reduced, and thinks that this indicates that a more **liberal diet** may be given without injury to the patients, and that the effect of a freer diet may be beneficial in improving nutrition.

M. Manges[2] describes 8 cases of his own in which **free diet** was given in typhoid fever, and discusses the results that others have had with this method of dieting. Manges believes that the cases do better with a soft diet than on strict milk diet. He does not believe that hemorrhage, perforation, or relapses are more common when this method is used.

R. W. Marsden[3] believes in a more **liberal diet** in typhoid fever than is usually used. He ordinarily gives soft foods, and even minced meats, unless there are decided contraindications. He thinks that in choosing a diet one should judge according to the individual, and not have a routine practice, and that the patient's appetite should be an important indication of diet, provided contraindications to free diet are not present.

C. Baümler,[4] in reporting upon his observations of the **bath treatment** of typhoid fever, insists that the effect of the bath can not be considered to be due purely to the influence upon the temperature; the effects are produced largely by the influence upon the circulation, the nervous system, and metabolism, and it can not be denied that the influence of the bath upon the circulation may result in some cases in a **severe strain upon the heart.** This should always be kept in mind, and the baths should not be used as if there were absolutely no possibility of evil effects. The sudden contraction of the peripheral vessels which occurs if the bath is frequently repeated may overstrain the degenerated heart. This effect, however, is not often observed, the effect upon the heart usually being good. The treatment of **1019 cases** showed a mortality of 9.32%. If those cases in which death was imminent when they were admitted to the hospital be deducted, and if cases with incurable tuberculosis be excluded, the deaths were only 7.9%—a marked reduction in mortality. It has been suggested that the cause of the lowering of the mortality rate in typhoid fever is the lessening of the severity of the disease owing to better hygienic conditions rather than the use of the bath. Baümler presents observations which show that the disease is apparently not decreasing in severity, and that the reduction in mortality is due to better treatment. Of the deaths in the cases reported, 18 were due to perforation, 7 to hemorrhage, 19 to bronchopneumonia, 6 to croupous pneumonia, 16 to general metastatic sepsis, 9 to failure of the heart. It is worthy of note that in 2 cases he observed **embolus of the pulmonary artery** as the cause of death. He considers the frequent occurrence of bronchopneumonia to be largely attributable to infection through the mouth and aspiration, and insists upon the importance of care of the mouth. As to the use of alcohol, he notes that both the typhoid toxin and alcohol caused vasomotor paralysis —a demonstration of the fact that alcohol must be used with great care. The vasomotor effect of alcohol is of value when used before and after the bath to prevent the vasoconstriction which occurs with the plunge, and the condition of the heart at times is such that alcohol must be used; but as cardiac stimulants Baümler particularly recommends musk and camphor.

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[1] Boston M. and S. Jour., Nov. 23, 1899.

[2] Med. Rec., Jan. 6, 1900.

[3] Lancet, Jan. 13, 1900.

[4] Deut. Arch. f. klin. Med., vol. LXVI, 1899, Festschrift.

E. Hirschfeld[1] describes the results which he has had from treating typhoid fever with the **tepid bath** (85° F.). This method has been much more successful than the use of colder baths. In 1899 there was a mortality of 3.4% in 120 cases which were treated with tepid bath, while the mortality with the colder bath in previous years had been about 7.5%. He finds that the temperature falls lower after the warmer bath; this he attributed to the fact that the tissues part with their heat more rapidly in the tepid bath because there is less initial contraction of the blood-vessels. There is also less shivering afterward, and shivering raises the temperature by lessening the skin circulation and by involuntary action of the muscles. He thinks that the cold baths also stimulate the production of heat to a much greater degree than the warm baths. In weak persons and in children he used the bath at a temperature of 90° F. He considers that only a tepid bath is permissible when the heart is very weak. If the patients are delirious and object to the cold baths, if they shiver much after cold baths, or if they greatly dislike them, he believes that the tepid baths will prove much more useful. [Judgment must be used in regulating the temperature of the bath as in every other detail of treatment. Patients react very differently to baths of a fixed grade of temperature, and the physician must be guided accordingly.]

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[1] Australas. M. Gaz., Jan. 20, 1900.

J. C. Wilson and J. L. Salinger[1] report the results of 10 years' experience in the treatment of typhoid fever by **systematic cold bathing, 1904 cases** being reported. The total mortality was 7.5 %—almost exactly identical with that of F. E. Hare in the Brisbane Hospital, Australia. The average mortality of the past 9 years in the city of Philadelphia has been 14.5 % ; and when compared with the mortality in the Brisbane Hospital for the 5 years preceding the use of the cold plunge, the general mortality of Philadelphia seems to be almost identical with this, which was 14.8 %. There has been an irregular decrease in the mortality in recent years in the general Philadelphia statistics, which the authors attribute, partly at least, to the fact that the Brand method is more generally used. The difference in mortality between those cases treated by the systematic cold bath and those cases treated by all methods in general is about 50 %. A number of series of cases at the German Hospital, taken at different times, show a mortality varying from no deaths in 40 cases to a maximum of 18.4 % in 27 cases, the general mortality being 7.42 %. The variations in mortality show that a large number of cases must be used to determine the value of the method. In summarizing the cases in the various hospitals in which the cases reported were treated, they note that in the German Hospital in 1898, with a total of 209 cases the mortality was 9.57 %. The Widal reaction done in the hospital laboratory gave 89 % of positive results, 11 % of negative results. The reaction as done in the city laboratory gave 94 % of positive results and 6 % of negative results. Albumin was present in over 84 % and nephritis was considered present in about 38 %. In the 52 cases examined the diazo reaction was present in 67.3 %. There were 267 cases treated in 1899, 231 of which gave positive Widal reaction in the hospital laboratory, while 188 were positive in the city laboratory. Albumin was seen 195 times. The diazo reaction was positive in 104 cases, negative in 64 cases. Relapse occurred in 22 cases. There was one case of combined malarial infection. From a study of the results they decided that cold bathing does not decrease the frequency of hemorrhage ; that it does seem to decrease the frequency of perforation. The danger of complications—particularly those of the respiratory and circulatory tracts—is lessened, there is apparently some increase in the frequency of relapse, and albuminuria occurs in a large percentage of cases with this treatment. At the German Hospital in the beginning of the attack purgatives were used, usually calomel, and external applications of cold were made. Medication is used only when some special complication indicates it. A bath is given every 3 hours when the maximum temperature reaches as high as 101.4° F. One or two plunges are given for some time even after the fever has subsided. The patients who are only slightly ill always walk to the tub and back to the bed. This theory is defended by a report of 776 cases treated in this manner, in which the mortality was only 7.8 %. It is suggested that it is useful to have the patients undertake muscular action, because enforced repose has an unfavorable effect upon nutrition; exercise increases the flow of the body fluids, and thus helps to get rid of the toxemia. [We would again record our objection to this practice, despite the figures offered in its support. The supposed advantages are doubtful and are not the primary reason for the adoption of the practice. Convenience alone suggested to the Germans, who first advocated it, the propriety of allowing patients to walk to the tub. The friction in the bath and the muscular exertion of shivering and increased respiratory action are far more powerful aids to the flow of body fluids than the walk to the tub.]

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[1] Phila. Med. Jour., Mar. 3, 1900.

L. Riess [1] insists upon the value of treating typhoid fever by **protracted baths.** While admitting that fever is not of itself a common cause of serious symptoms, he believes that the height of the fever usually indicates the severity of the disease, and that improvement occurs when the fever is controlled. When protracted baths were used, the duration of the disease seemed to be shortened, the mortality reduced, and the complications lessened in number. The average duration of 809 cases was 17.7 days; the mortality, 8.5%.

E. Drocker [2] speaks highly of **constant sponging** in reducing temperature. [We can fully agree with the author as to the usefulness of this procedure, having frequently found it more effective than either packs or the plunge bath in typhoid fever, as well as in other conditions when the temperature persistently tends to remain high. It is well, however, to recognize that the plunge is a more powerful roborant even in cases in which it does not reduce the temperature so much as a prolonged sponge-bath.]

D. Rochester,[3] in discussing the **local nonsurgical treatment of the intestine** in typhoid fever, states that his results from the use of benzoate of guaiacol have been unfavorable, and that he does not feel that the use of drugs for intestinal disinfection will have much effect. He also has had unfavorable results from the use of more liberal diet than milk and other liquid foods, and feels safe only when using milk, usually peptonized. If vomiting occurs, he stops the food for half a day or a day and uses cerium oxalate and calomel. He prescribes abundant amounts of distilled water in typhoid, and advocates the regular use of calomel in laxative doses every 4 or 6 days during the course of the fever, together with flushings of the colon with from 2 to 5 liters of water.

B. M. Taylor,[4] in considering the **treatment of typhoid fever,** makes a division between those patients who were in good health before acquiring typhoid fever and those who had had chronic disease for some time before. In the first class he makes the interval between administrations of food longer than usual in order to avoid overtaxing the stomach. He discountenances the regular use of stimulants and disapproves of purgatives. He gives a freer diet than is usually advised. He advises irrigation of the colon 2 or 3 times a day and sponging the body with warm water. He considers it proper to use coal-tar preparations to reduce the temperature. The matter of importance in cases with chronic disease he considers to be careful attention to the condition of the stomach.

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[1] Deut. med. Woch., Oct. 5, 1899.

[2] National Hosp. Reports, Sept., 1899.

[3] Phila. Med. Jour., Nov. 4, 1899.

[4] Med. Rec., Sept. 16, 1899.

E. Speidel [1] **in treating typhoid fever** recommends the evacuation of the gastro-intestinal tract and the prohibition of food for 5 or 6 days in order to further asepsis of the whole canal as far as possible. Also, water should be given regularly, and he advises rectal irrigation daily, except in hemorrhage. He considers that buttermilk is the best food for such cases, but advises some variation in diet in order to tempt the patient to take plenty of food.

J. H. Musser,[2] in discussing the indications for the **use of alcohol** in typhoid fever, divides the cases into 2 general classes. In one in which toxemia is manifested by excitement stimulants are to be avoided, and the one valuable drug is opium; when there is marked adynamia with dullness, dry tongue, dicrotic pulse, and scanty secretion, stimulants must often be used in large quantities. The best indication of the effect of stimulation is in the amount of urine. If as much as 50 ounces is secreted, Musser feels free from anxiety.

E. C. Seufert [3] reports 33 consecutive cases of typhoid fever with no deaths. All the cases were given antiseptics, beginning with calomel and salol and following these with naphtha and guaiacol carbonate. The average duration of the disease was 21 days.

G. F. Butler [4] recommends guaiamar, which is a product of guaiacol and anhydrous glycerin, as an intestinal antiseptic. He believes that he has had excellent results from its use in 20 cases of typhoid fever. It is said to increase the appetite and to improve digestion, not irritating the stomach. It should be given in doses as large as 10 to 15 grains 3 times a day if it causes no irritation of the genito-urinary tract. As an external ointment he has found that it lessens the pains in rheumatism.

C. R. Carpenter [5] uses **splenic extract** in the treatment of typhoid fever and other infectious diseases unaccompanied by leukocytosis, because he believes that leukocytosis is an active factor in overcoming infection, and splenic extract has repeatedly been shown to cause a leukocytosis.

M. Hougeletk [6] gives a general consideration of the treatment of typhoid fever by **enteroclysis.** He considers this method quite as satisfactory as the bath treatment, and thinks that the patients convalesce more rapidly than when baths are used. It also increases the excretion of urine, and, he believes, aids in eliminating the toxins from the system.

A. E. Wright and W. B. Leishman [7] discuss the preparation of the **typhoid serum,** and report the results obtained by Wright in some work on soldiers in India. The vaccine used was made from 4 weeks' old cultures of virulent typhoid bacilli to which lysol was added, the vaccine having been prepared a year before its use. This was employed in a dose of 0.5 cc. to 0.7 cc. Cultures sterilized at 60° C. were also used. These were 24-hour cultures on nutrient agar, and were given in doses from 0.3 cc. to 0.5 cc. The use of either was followed by decided reaction. After studying the instances of typhoid in the persons inoculated and comparing them with the records in troops which were not inoculated, it was decided that the vaccine conferred a very definite degree of immunity upon the persons so treated.

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[1] N. Y. Med. Jour., Dec. 30, 1899.
[2] Therap. Gaz., April 15, 1900.
[3] N. Y. Med. Jour., Aug. 12, 1899.
[4] N. Y. Med. Jour., Sept. 23, 1899.
[5] Med. Rec., Feb. 17, 1900.
[6] Thèse de Lyon, 1899.
[7] Brit. Med. Jour., Jan. 20, 1900; and Lancet, Jan. 20, 1900.

R. W. Marsden[1] reports that he has carried out Wright's **preventive inoculations** with the typhoid serum in 14 of 22 nurses who were engaged in the hospital of which he is superintendent. Four of the other nurses had had typhoid fever and the remaining 4 did not wish to submit to inoculation. While previously for the past 5 years there had been from 3 to 8 cases of typhoid among the nurses, and from January to September, 1899, 5 cases had occurred, from September, 1899, to March, 1900,—the period during which the inoculations were carried out,—no cases occurred, unless one obscure case might be considered typhoid fever. If this were typhoid fever, it was so evanescent and mild that Marsden considers that it would be excellent testimony for the protective action of the inoculations.

D. Duckworth[2] reports his use of antityphoid inoculation in a young man who was about to go on duty in the tropics. He gave 2 injections of 1 cc. of antityphoid serum 10 days apart. After each injection there was some fever, anorexia, and a little pain and redness about the point of injection. After the inoculation the Widal reaction was marked in a dilution of 1 : 200. Another patient was inoculated, and similar symptoms followed. Duckworth believes that a satisfactory result may be obtained from these inoculations if 2 are made, the second being given about 10 days after the first. The patient should be kept in bed for 2 days until the reaction is past.

T. Wilson[3] states indefinitely that he has used the **antityphoid serum** of Wright on the soldiers of South Africa, and he considers that it will either render the typhoid bacillus innocuous or will favorably modify an attack of typhoid fever. The number inoculated is not stated, but it evidently was large, since there is a statement that there were about 40 inoculated daily. Two cases of some collateral interest are reported. One was a case of acute gonorrhea. The discharge disappeared entirely the day of the inoculation, and the man was well of the inoculation within 3 days. In another case chronic gleet, which had been resistant to treatment, subsided at once after an antityphoid inoculation, but a debauch 2 days later caused it to return.

J. W. Washbourn,[4] in discussing **enteric fever** as it occurred in South Africa in the soldiers, stated that he did not think that inoculation had any decided effect in lessening the severity of the attacks; he describes 2 fatal cases in persons who had been inoculated. There were several instances of the occurrence of typhoid fever after dysentery.

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[1] Brit. Med. Jour., April 28, 1900.

[2] Brit. Med. Jour., Nov. 18, 1899.

[3] Brit. Med. Jour., April 28, 1900.

[4] Brit. Med. Jour., June 16, 1900.

T. R. J. Cowen [1] reports his use of **antityphoid serum** in a severe case of typhoid fever. The serum was given in 2 doses—one in the fifth week in a quantity of 3 cc., and a few days later 7 cc. As has been noted before, the temperature rose rapidly after both injections, and there was then a decided and rapid improvement in the general condition. The quickness of improvement suggested that the serum itself contained an antitoxin or that it caused rapid formation of one. After the injection there was difficulty of speech, and a convulsion occurred subsequently. These, which were attributed to embolism in the cerebral artery, left some permanent mental defect.

B. Boskett [2] describes a case which he had diagnosed as typhoid fever in which he used **antityphoid serum.** The patient soon improved, and Boskett believes that it was an instance of abortion of typhoid fever in the early stage. Subsequent trial of the Widal reaction was negative, which Boskett believes was due to the fact that the disease was aborted early. He does not think that this is sufficient to arouse question of the diagnosis.

## MALARIAL FEVER.

**Etiology.**—R. Koch [3] contributes a report of the Commission for the Study of **Malaria in Italy.** The most important point in the paper is perhaps his insistence that malaria is transmitted from year to year by the cases which relapse, and which harbor the parasite throughout the winter. It is well determined already that the plasmodium can not live in the mosquito except in the warm atmosphere, and there is no knowledge that the plasmodium can live in any outside host except the mosquito; therefore Koch believes that the disease must be perpetuated by the cases that relapse, and that these cases should be treated with energy and persistence, and should be looked upon somewhat as isolated cases of cholera and other epidemic diseases are looked upon. In other studies it was found, as he has noted before, that the estivo-autumnal form of the fever began as the tertian type and became irregular when some immunity had been conferred by quinin. In all, 281 cases were treated at the hospital and outside; in the tertian form by giving 1 gm. of quinin twice, repeating this dose every 2 or 3 days for a week or two, and subsequently every 10 days. In the tropical form the quinin was given only when the large ring-shaped parasites appeared. One case of hemoglobinuria was seen. It appeared after a large dose of quinin when the man had been accustomed to taking small quantities of the drug. A similar attack had occurred in the same patient when he was 4 years old, and also came on after a large dose of quinin. In considering the relation of the various mosquitoes to malaria, Koch reports that the Commission believes that the Culex pipiens and the Anopheles maculipennis are both active in spreading the disease. Coccidia-like parasites were found in both forms. He believes that it is entirely improper to criticize the theory of the mosquito transmission of malaria with the argument that the mosquito is found throughout the winter while malaria is not. In answer to this, he cites the fact that analogous work with the proteosoma shows the necessity for a certain degree of warmth before the parasite can develop, and it will be found upon careful observation that just about this degree of warmth is present in houses at the time that malaria breaks out in the spring and early summer.

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[1] Lancet, Sept. 16, 1899.

[2] Brit. Med. Jour., Sept. 30, 1899.

[3] Deut. med. Woch., Sept. 14, 1899.

W. F. Arnold[1] reports an interesting observation concerning the occurrence of severe Cuban malaria in the United States navy during the war with Spain. About one-third of the severe attacks in the navy were seen on 3 gunboats—the "Manning," the "Vixen," and the "Scorpion." In the cases on the "Vixen" and the "Scorpion" the infection might have been considered due to drinking-water, but in the case of the "Manning" this could not have occurred, as no drinking-water from the shore was used. The **only possibility of infection was from mosquitoes** when the "Manning" was lying about 300 meters from the shore. She never went alongside the pier at Daiquiri, where the infection occurred; and in the absence of the possibility of infection by drinking-water, the only source of infection that seemed at all reasonable was mosquitoes.

T. Smith,[2] in discussing the **etiology of Texas cattle-fever** with special reference to recent hypotheses concerning the transmission of malaria, directs especial attention to the fact that the parasite of Texas fever often persists in the blood for a long time after the animals recover, and that in this way opportunity is afforded for the infection of animals that would otherwise escape. From analogy with this he considers that persons infected with malaria, but showing no active evidences of the disease, and therefore not undergoing treatment, are likely to cause infection of others, particularly in regions that are not already infected. Such cases do grave damage by perpetuating malaria.

A. E. Woldert[3] reports some histologic studies of the mosquito which he has undertaken preliminary to further investigations relating to the **inoculation of malarial fever by the mosquito.**

J. Reid[4] advises that **mosquitoes** and similar insects be **mounted in glycerin** in examining them. He allows them to become entangled in the glycerin, and uses fine needles to place them in proper position. In this way they are not likely to be injured, air-bubbles do not form, and they may be advantageously studied.

R. Ross[5] reports a series of cases of malaria in which it is believed that there was sufficient **evidence of inoculation by mosquitoes.** The facts concerning the cases were related to Ross by a layman, but were given accurately. A company of a boys' brigade, consisting of 13 boys, was in camp near Calcutta, and all 13 boys, a sister of one of the boys who visited the camp, and 3 servants had malaria after settling in the camp. These persons were not protected by mosquito nets, while 3 officers and others who used nets had no malaria.

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[1] Phila. Med. Jour., April 7, 1900.

[2] N. Y. Med. Jour., July 8, 1899.

[3] Jour. Am. Med. Assoc., Feb. 3 and 10, 1900.

[4] Brit. Med. Jour., Jan. 30, 1900.

[5] Brit. Med. Jour., April 22, 1899.

I. Macdonald [1] has studied **malaria in the Spanish Sierra** in its relation to mosquitoes. He decides that **Anopheles claviger** is the mosquito most frequently found in this region, and he thinks that it is therefore the one chiefly active in propagating malaria. W. S. Thayer [2] found that in Baltimore proper, where malaria is rare, the anopheles was uncommon; while in the surrounding districts, where malaria is prevalent, Anopheles quadrimaculatus was frequently found. He found also that the anopheles was present in Jackson, N. C., in Newport News, and in the swampy districts surrounding New Orleans, in which situations malaria occurs frequently. W. N. Berkley [3] considers that the anopheles is the most frequent transmitter of malaria, if not the only mosquito that is active in the transmission of the disease. He searched for this form of mosquito in New York during the autumn of 1899, but did not find it. He did, however, discover several specimens of culex, but was unable to get any positive results in his experiments concerning its position as a host for the malarial plasmodium. He insists that malarial patients should be isolated from mosquitoes and should be considered a source of danger to surrounding persons.

H. Ziemann [4] states that after long search he was able to find **mosquitoes in Kamerun** which showed infection with malarial organisms. He discovered in the stomach pigmented coccidia-like bodies which became transformed into the so-called sporozoids and finally reached the salivary glands. J. B. van Berlekom [5] reports an epidemic of malaria in Zeeland. Previous to the summer of 1899 malaria had occurred in this province only in sporadic cases. Last summer an epidemic appeared in Middleburg. Berlekom attributes the epidemic to bad hygienic arrangements and to fouling of the soil. The malarial parasite was constantly found. He believes that mosquitoes had nothing to do with the epidemic.

**The Plasmodium.**—C. F. Craig [6] considers that there are **two forms of flagellated malarial organisms** which appear in the blood in tertian and in estivo-autumnal fever, and that they are probably 2 separate varieties. In order to obtain flagellated bodies he recommends that after puncture a well-cleansed slide should be breathed upon gently, and after moistening in this way touched to the drop, and the cover-glass immediately placed upon it. The slight exposure of the blood and the moistening of the slide he believes hasten the appearance of the flagellates, and specimens so prepared usually contain these bodies. He calls the 2 forms the active and the passive, the active being most common in tertian fever. Its characteristics are chiefly the extreme activity of the pigment, the appearance of the clubbed extremity of the flagellum, and the separation and individual existence of the flagella and their power of movement. The passive form is more common in estivo-autumnal fever, and is found, in Craig's experience, only in those cases in which crescents are present; he considers that they develop from crescents. In this form the pigment is not active, there is no clubbed outer extremity, but there is a nodule at the junction of the flagellum with the body. This form has a rapid revolving movement upon its axis and often becomes loosened from the body and again attaches itself to it, while in the active form the flagella have serpentine lashing movements.

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[1] Brit. Med. Jour., Sept. 16, 1899.

[2] Phila. Med. Jour., May 5, 1900.

[3] Med. Rec., Dec. 23, 1899.

[4] Deut. med. Woch., June 21, 1900.

[5] Nederl Tidsch. for Geneeskunde, Nos. 6 to 8, 1900.

[6] N. Y. Med. Jour., Dec. 23, 1899.

C. S. Engel [1] discusses the **possibility of confusing** some forms of **nucleated red corpuscles with malarial plasmodia.** He decides that plasmodia of the older form, those containing yellowish-black pigment, could scarcely be confused with nucleated red cells; neither could those with ameboid movement. Plasmodia also are not readily confused with polychromatic normoblasts, since the malarial organisms are found almost exclusively in orthochromatic red cells. There is little danger of confusion of the plasmodia with orthochromatic nucleated red cells, for the nuclei of the latter stain intensely, while the stain taken by the plasmodia is much less marked. Engel considers, however, that there is distinct danger of confusing plasmodia with the forms of nucleated red cells in which the nucleus is broken up into fine particles and those in which the nucleus gradually becomes smaller and ultimately disappears. The blood plaques may also be readily confused with plasmodia spores.

A. E. Woldert [2] describes a **method of staining blood** for the demonstration of the malaria plasmodium. It consists in the use of 3 solutions, as follows: (I) toluidin-blue 15 grains, distilled water 4 drams; (II) acid fuchsin 15 grains, distilled water ½ dram; (III) 2% watery solution of eosin. To No. I add 20 drops of No. II, and afterward 20 drops of No. III. A precipitate occurs which should be left in the bottle. It stains the parasites blue, the cells pink, and the nuclei of the leukocytes blue.

**Symptomatology.**—S. Kanellis and J. Cardamatis [3] discuss the **relation between pernicious malaria and dysentery.** They have had a large experience themselves, having seen about 10,000 cases of malaria in Greece, and have collected statistics of about 277,000 cases; of these, 3054 were pernicious malarial fever. In only 8 of the latter cases was the malaria associated with dysentery, and these 8 cases occurred in countries where dysentery is quite as epidemic as malaria. They admit that malarial fever and dysentery are often associated in ordinary cases, the anemia and general depression of the malarial subjects making them susceptible to dysentery; but they consider the two diseases distinct, and do not think that malaria itself produces dysenteric symptoms. One point in which error is likely to arise is that there may be marked fever in dysentery; and if this occurs in malarial regions, it is often considered to be a malarial fever.

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[1] Zeit. f. klin. Med., Bd. XXXVIII, Hefte 1, 2, u. 3.

[2] Phila. Med. Jour., April 14, 1900.

[3] Bull. Acad. méd., Jan. 9, 1900.

D. C. Rees [1] reports a fatal case of **malignant malarial fever** which was seen in England, and occurred in a man who had just come from Africa. Enormous numbers of parasites were found in the blood, many corpuscles containing as many as 4 parasites, which were chiefly of the small, ameboid, nonpigmented form. Ten grains of the hydrochlorate of quinin were given every 4 hours without any definite effect. Pigmented lymphocytes appeared in the blood, and some cells which looked like myelocytes and which contained an enormous amount of pigment. Hemoglobinuria was absent. The postmortem showed extreme softness of the spleen; the vessels of the brain were much congested, and held large numbers of red corpuscles containing parasites and numerous leukocytes holding masses of pigment. The liver and spleen were much pigmented.

B. M. Taylor [2] records the case of a child who suddenly became **wildly delirious,** with a slightly **subnormal temperature** and slow respiration. He then became entirely motionless. The temperature was subnormal for 7 days, with a pulse of 65, the slow respiration and stupor continuing. On the fifth day Taylor saw him and discovered malarial plasmodia in his blood, and after administering large doses of quinin hypodermically, the child recovered and the plasmodia disappeared entirely from the blood.

C. W. Larned [3] reports a case which he considers **chronic malarial nephritis.** The patient was a 7-year-old child that was suffering from quartan malaria and had a nephritis which was believed to be due to an existing malaria or to previous attacks of the disease. The child died in convulsions. Larned thinks that it is important that blood examinations for the malarial organisms should be made very frequently in case of nephritis, particularly when the patients have been living in a malarial district, as the clinical picture may be apparently that of a nephritis. [The relation of malaria to nephritis is very uncertain, though it is not unlikely that the renal lesion sometimes results from malaria.]

R. F. Austin [4] describes a case of **hemoglobinuria** in a patient with malaria. Recovery occurred after the administration of large doses of quinin subcutaneously for several days. There was marked albuminuria persisting for some time after the blood disappeared.

W. H. Crosse,[5] from investigations on the **histology of blackwater fever,** concludes that the appearances are not different from those found in the tissues of persons who have been subjects of malaria. He believes that blackwater fever is an intoxication of malarial origin. The prevention of it he considers is chiefly avoidance of infection through mosquitoes and the administration daily of 5 grains of quinin. F. Smith [6] describes a case of blackwater fever which occurred in a patient who had for 10 months taken 10 grains of quinin every other day. Hemoglobin appeared on the third day of the fever, and the following day examination of the blood showed a quartan malarial parasite in small numbers.

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[1] Brit. Med. Jour., Feb. 1, 1900.
[2] Med. Rec., Dec. 2, 1899.
[3] Johns Hopkins Hosp. Bull., July, 1899.
[4] Brit. Med. Jour., Feb. 1, 1900.
[5] Lancet, Jan. 6, 1900.
[6] Lancet, Nov. 4, 1899.

W. Osler [1] describes a case of **gangrene in malarial fever.** The man was 23 years of age, and had had repeated attacks of malarial fever. The attack described began with what was thought to be influenza, but was probably malaria. Blebs appeared on the hands, and subsequently mottled areas appeared elsewhere on the skin surface and about the blebs, and there was a rapid development of localized gangrene. The blood was found to contain large numbers of malarial crescents. Blood cultures were negative and there was no leukocytosis. The man recovered entirely.

**Treatment (Prophylaxis).**—R. Ross,[2] in considering the question of the **extermination of malaria,** suggests the possibility that this may be accomplished by exterminating the mosquito. He believes that Anopheles claviger is the one that conveys the infection in tertian fever. He notes that when this insect is young it lives entirely in the water and takes about a week to mature. It would then be possible to destroy all the mosquitoes in a given region if all the water found in ditches, puddles, and the like in the neighborhood were emptied out as often as once a week. Proceeding upon this plan, a method of preventing malaria will be found.

I. P. Lyon [3] presents an interesting critical review of the literature concerning the inoculation of malaria by the mosquito. He ends by stating that every case of malaria may become a **menace to the public health** if the anopheles exists in the same locality. Such cases constitute foci of infection, and Lyon believes that the periodic appearance of malaria in portions of the country that are usually free from the disease is explained by this method of infection of human beings. He thinks that active cases of malaria should be isolated by mosquito netting or by some other method. The best method of killing the mosquitoes is by draining the pools of the neighborhood or by covering their surfaces with petroleum or some other chemical. One ounce of petroleum to 15 square feet of water will destroy the mosquito grubs and will probably prevent their development for from 2 to 4 weeks.

M. Lavaran,[4] in speaking of the transmission and prophylaxis of malaria, recommended the use of petroleum spread on the surface of water in the destruction of mosquitoes, but stated that even better results were obtained with **fresh tar poured on the water** drop by drop. This has the advantage of evaporating less rapidly than petroleum. [Proper drainage and the avoidance of pools or swamps are the measures that will prove most useful in eliminating malaria. Destruction of the larval insect is much less likely to prove successful.]

C. Celli,[5] in a discussion of malaria and its prophylaxis, states that the first point in prophylaxis is **isolation of the patient.** One of the most important steps is disinfection of the patient himself and destruction of mosquitoes. The useful methods of destroying mosquitoes are the use of petroleum, anilin dyes, and Dalmatian chrysanthemum flowers. They are best destroyed in the winter and spring. It may be remembered that for every mosquito killed there will be from 200,000,000 to 20,000,000,000 less the following year. The recurrence of malaria should be prevented by avoidance of sleeping in the open air or of exposing one's self freely to the evening and early morning air, the avoidance of mosquitoes, protecting one's self from these insects, and the construction of proper sewerage systems. Proper cultivation of the soil is very important, preferably by dry methods; if irrigation is used, the fall of water should be made considerable. Methylene-blue may be taken internally by persons disposed to malaria to prevent the occurrence of the disease.

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[1] Johns Hopkins Hosp. Bull., Feb., 1900.

[2] Brit. Med. Jour., July 1, 1899.

[3] Med. Rec., Feb. 17, 1900.

[4] Bull. Acad. de méd., April 3, 1900.

[5] Berl. klin. Woch., Feb. 5 and 12, 1900.

E. J. E. Risk [1] uses hypodermically 10-grain doses of quinin dissolved in tartaric acid and water in the **grave forms of malaria** which he sees in St. Lucia. The vomiting often interferes with medication by the mouth, and rectal medication is a necessity. The common remittent fever of St. Lucia shows in the blood the estivo-autumnal parasite, often in large numbers.

Smithwick [2] describes 50 cases of malaria of different kinds which he has treated with **methylene-blue** with satisfactory results. He finds that this drug may be used in the place of quinin when the latter can not be administered. He thinks that there are no idiosyncrasies to methylene-blue, and that it is valuable in the hemorrhagic type of malaria because it is a diuretic as well as a parasiticide. It may also be used in pregnancy, as it has no oxytocic effect. M. Moussoos [3] has observed within 10 years 60 cases of bilious hemoglobinuric fever resulting from malaria. He gives in this condition **subcutaneous injections of methylene-blue** with success. He does not consider the condition described due to quinin.

A. O. Fitzgerald [4] has treated a series of cases of malaria by **inunctions** of **creasote** in equal quantities of olive oil, the dose used for adults being from 30 mm. to 60 mm.; for children, 15 mm. to 20 mm. He thinks that a specific effect was observed, and that it is an extremely satisfactory method of treatment.

## INFLUENZA.

H. S. Anders [5] has studied the **relation of the local meteorologic conditions** to the influenza epidemic in Philadelphia in the winter of 1898–99. He decides that there were several volleys of attack, during which for several days large numbers of people became affected by the disease. The disease then subsided partly, but exacerbations were seen afterward; the exacerbations being preceded always by foggy, moist, and relatively calm weather, while the onsets of the exacerbations were coincident with clear, dry, and windy weather. This suggests that the weather conditions may be related to the occurrence of epidemics or pandemics, influencing either the susceptibility of the persons affected or the virulence of the micro-organisms. [As has been the case in former times, the great waves of pandemic influenza have been followed by endemic and more or less epidemic forms of irregular and often obscure type.]

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[1] Brit. Med. Jour., Nov. 25, 1899.
[2] Merck's Archives, Feb., 1900.
[3] Bull. Acad. de méd., Oct. 10, 1899.
[4] Brit. Med. Jour., July 15, 1899.
[5] Phila. Med. Jour., Aug. 19, 1899.

N. Filatow [1] discusses the **protracted chronic forms of influenza.** These are chiefly evidenced by the occurrence of protracted fever without intermission or by severe repeated attacks of influenza which recur at intervals for months or years. In the continued forms the fever lasts from 6 weeks to 3 months, or longer. There are usually no marked rises in the fever, but in the afternoon particularly the patients are likely to complain of the usual subjective symptoms associated with chills. There are often no catarrhal symptoms in these chronic forms. The conditions with which this form of the disease is likely to be confused are typhoid fever, malaria, and miliary tuberculosis. Typhoid fever and malaria may usually be excluded by laboratory methods of diagnosis. As to tuberculosis, one of the most important points is that chronic influenza is likely to affect several members of the family at the same time; also the temperature does not tend to go high and does not remit markedly. There is danger of complication with tuberculosis, however, in chronic influenza.

Huchard [2] discusses the **attenuated forms of grip.** He first mentions the apyretic varieties, in which there may be marked pulmonary congestion or actual lobar pneumonia without fever. The striking point about these cases is that they advance insidiously without fever, without expectoration, and often without cough. [Sometimes cases of this sort, though as a rule afebrile, are liable to occasional elevations of temperature, arising without definite cause.] The condition is often discovered only upon auscultation. Under the febrile forms of attenuated grip he describes cases in which the disease is likely to run a very rapid course, but in which, with severe fever, there are no discoverable anatomic lesions. He directs especial attention to the ambulatory forms of grip, which are of great importance because they have so much to do with the spread of the disease. He insists that it is necessary in all cases of grip to prevent infection of others as far as possible, and the point upon which he especially insists is careful disinfection of the mouth cavity and of the skin surface. In the treatment of the disease he finds sulphate of quinin, particularly when combined with ergot, most valuable, and utters a warning against the free use of antipyrin and similar preparations, because of the danger of heart failure.

R. J. Colenso, [3] in discussing **influenza of the gastric type,** describes it as beginning usually with some coryza and bronchial irritation, together with irritation of the mucous membrane of the alimentary tract, causing profuse redness and swelling of the pharynx and marked disturbance of the stomach. The spleen and liver usually enlarge, and there may be icterus. At first there is constipation, and later diarrhea; there is often distinct resemblance to enteric fever, sometimes to dysentery, or even to cholera; skin eruptions are frequent. Following these attacks there are likely to be gastro-enteric sequelæ, such as atonic dyspepsia, and there is frequently a dry cough from pneumogastric irritation.

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[1] Deut. Nediz. Zeitung, Aug. 28, 1899.
[2] Bull. Acad. de méd., Feb. 17, 1900.
[3] Practitioner, Aug., 1899.

R. Lee [1] believes that the pain which is frequently felt about the base of the chest in influenza is due to **involvement of the diaphragm,** and he considers that spasm of the diaphragm produces the dry spasmodic cough so often seen. He likewise believes that spasm of the diaphragm may cause death in some obscure cases of sudden dissolution.

B. J. Byrne [2] discusses **nervous depression as a sequel** of influenza, directing especial attention to the occurrence of subnormal temperature and bradycardia as indications of nerve depression. He treats the condition chiefly by large doses of strychnin with brandy, insisting upon complete rest. He often uses the cold bath.

A. D. Rockwell [3] describes a case of **dyspnea following grip,** which was believed to be due to paralysis of the diaphragm, since the epigastrium became depressed in inspiration and became full in expiration. The case was cured by the use of faradism, later combined with galvanism. The use of the two currents together is believed to be better than one alone. Rockwell believes that electricity is valuable in respiratory failure, but not advisable in cardiac failure.

J. H. Spitzly [4] reports the case of a man of 25 who was taken with a severe attack of influenza, in which there was decided delirium and high fever. On the fourth day, with a drop in the temperature, violent **maniacal delirium** developed, and continued in greater or less degree for a week. There were no local complications, such as pneumonia. Since the delirium appeared with the influenza and disappeared as it grew better, it was believed that it was due to influenza poisoning.

M. F. Austin [5] describes 3 cases of **endocarditis** in which the examination of the valves of the heart showed the presence of micro-organisms with the characteristics of the **influenza bacillus.** It was thought that the endocarditis was probably due to direct infection with the bacillus of influenza. [This observation may be accurate, but can not be accepted unreservedly. It is certain that influenza does not often cause endocarditis.]

J. E. Hermann [6] has frequently observed **eruptions resembling scarlet fever, measles, and herpes** in influenza. He thinks that the epidemic of intercostal neuralgia with herpes reported by Reilly was really influenza with herpes. Similar cases seen by Hermann showed the presence of the Pfeiffer micro-organism. [Herpes is recognized as a frequent attendant of influenza.]

Rieger [7] describes 3 cases of influenza in which there occurred a skin

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[1] Lancet, Jan. 27, 1900.

[2] Jour. Am. Med. Assoc., Mar. 10, 1900.

[3] Med. Rec., Nov. 11, 1899.

[4] Brit. Med. Jour., Mar. 3, 1900, p. 508.

[5] Johns Hopkins Hosp. Bull., Oct., 1899.

[6] N. Y. Med. Jour., Feb. 17, 1900.

[7] Münch. med. Woch., Jan. 2, 1900.

**eruption** somewhat **like syphilis.** He suggests that the eruption was an attempt to eliminate the influenza poison.

De la Mallerée [1] recommends the **treatment** of influenza **by isolating patients** in a room and giving them inhalations of formaldehyd. The result is said to be rapid disappearance of the cough, decrease in the fever, and disappearance of the influenza bacilli from the sputum within 48 hours. After 48 hours have passed the patients are placed in a second isolated room, but the inhalations are stopped. No bad results were seen from this treatment.

Burgen [2] reports 17 cases of influenza which he treated by the **open-air method,** windows and ventilators being left wide open through the 24 hours, the temperature in the wards being only slightly above that without, and averaging about 42° F. He believes that the range of fever was lessened, that the complications were fewer, and that the fever was of shorter duration. [We have always feared exposure in this disease, having repeatedly observed unfortunate complications after very slight exposure. The treatment suggested by Burgen does not, therefore, seem to us free from danger.]

## TYPHUS FEVER.

A. Balfour and C. Porter [3] describe the **bacteriologic study** of 143 cases of typhus fever. They obtained blood from the thumb in all cases after very careful antiseptic precautions. In 88% they found a diplococcus, the cultural and morphologic characteristics of which they describe at length. They found the same organism in 87% of 46 cases of typhoid fever, all of which, with one exception, gave the Widal reaction. They also found it postmortem in cases of typhoid fever. They did not find the organism in other diseases investigated or in normal persons.

H. Littlejohn and C. B. Ker [4] describe an **epidemic of typhus fever** which occurred in Edinburgh in which 82 cases were seen. The death-rate was 12%. One-fourth of the cases occurred in children under 10; no deaths occurred in patients under 15. Almost all the cases occurred within a limited radius and in a district in which the hygienic arrangements were extremely bad. The average incubation period was about 2 weeks. They noted that the subcuticular element of the rash usually appeared as early as the third day. Death rarely occurred before the end of the second week, and in nonfatal cases the temperature usually became normal by the fifteenth day. Delirium was very common in adults, particularly in the fatal cases. Albuminuria was present in 38 of the 51 adult cases. The diazo reaction was seen in each of the 15 cases examined for it. The tendency was at first to constipation, later to diarrhea, which became uncontrollable if purgatives were given. One patient was seen in a second attack of typhus. Among the notable complications were otorrhea, severe diarrhea, and furunculosis. Most of the fatal cases occurred in large, muscular men ; nearly all of these had been alcoholic. The authors think that a good physique is a disadvantage in typhus fever. A profuse rash indicated a grave attack. The Widal reaction was a valuable method of diagnosis from typhoid fever, and the rotten-straw odor was considered a useful indication of typhus. None of the attendants upon the patients contracted the disease, owing, the authors think, to the free ventilation and careful hygienic regulations.

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[1] Bull. Acad. de méd., May 29, 1900.

[2] Brit. Med. Jour., June 30, 1900.

[3] Edinb. Med. Jour., Dec., 1899.

[4] Edinb. Med. Jour., July, 1899.

## CEREBROSPINAL FEVER.

**Etiology.**—H. Jaeger [1] thinks it is proved that epidemic cerebrospinal meningitis is **due to the meningococcus.** He notes that he has found this organism in 17 cases since his earlier publications. He thinks the differentiation from the pneumococcus is easy, but that it is much more difficult to distinguish from the staphylococcus. Hünermann's results he attributes to a confusion of the staphylococcus with the meningococcus. He considers it of importance that the meningococcus grows but slowly, if at all, upon gelatin, and does not liquefy the gelatin, while the staphylococcus liquefies it rapidly. The meningococcus is a wide-spread organism and is ubiquitous, and this explains the occurrence of sporadic cases, while the fact that epidemics occur only occasionally is due to the lack in human beings of marked susceptibility to the disease. That the meningococcus will resist long periods of drying is shown by the fact that Jaeger was able to transplant the meningococcus from cultures that had been dried for as long as 96 days.

Hünermann,[2] in reply to Jaeger's criticism on the epidemic of cerebrospinal meningitis at Mayence, in which Jaeger stated his belief that Hünermann had confused the meningococcus and staphylococcus, insists that there is **no proof that all organisms described as meningococci are identical.** He also emphasizes the fact that epidemics may be caused by organisms other than the meningococcus, giving numerous proofs of this statement. He likewise notes that Jaeger considers that the recent epidemics in Germany have been due to infection from France, and he quotes Netter's statistics concerning the bacteriologic findings in 21 cases in Paris. The pneumococcus was found 7 times, the meningococcus 6 times, the streptococcus form of the meningococcus 4 times, the streptococcus pyogenes 3 times, and the staphylococcus once. Admitting Jaeger's contention that the source of infection was France, the bacteriologic results in Paris do not bear out his theory that the meningococcus is the constant cause. Fraenkel, too, has twice discovered the influenza bacillus. Hünermann insists that the disease can not be attributed solely to the meningococcus unless cultures show the absence of other organisms.

E. Stadelmann [3] reports the case of a man of 26, a subject of cerebrospinal meningitis, in which a **peculiar micro-organism** was found.

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[1] Deut. med. Woch., July 20, 1899.

[2] Deut med. Woch., Sept. 28, 1899.

[3] Deut. med. Woch., July 20, 1899.

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The cultural characteristics are described. The chief peculiarity was that the micro-organism developed only after the culture had been kept in the incubator for a week. Stadelmann is convinced that epidemic cerebrospinal meningitis may be due to either the meningococcus or the pneumococcus, and probably to other micro-organisms. He considers it proved, at any rate, that epidemics of cerebrospinal meningitis have been caused by the pneumococcus, and believes that it is only to be expected that more than one micro-organism should cause this affection, since other diseases sometimes epidemic, particularly pneumonia, may be due to various organisms.

L. Zupnik [1] reports a case of cerebrospinal meningitis of the epidemic form which ended fatally. Lumbar puncture during life, as well as postmortem bacteriologic studies, showed the presence of a diplococcus, which morphologically seemed to be the meningococcus, but which did not grow upon bouillon, blood-serum, or glycerin-glucose-agar, and only very slightly upon agar. It therefore seemed that it was **not the meningococcus,** and its nature remained in doubt, as no other cultures were instituted, since at the time cultures were made it was supposed that it was the meningococcus.

W. J. Buchanan [2] reports a case of cerebrospinal fever in India in a man of 52. Lumbar puncture was carried out and the **diplococcus** was found. The man died, and the postmortem showed a leptomeningitis of the surface of the brain with a collection of lymph upon the base, medulla, and upper part of the cord. Diplococci were found in the exudate.

**Symptomatology.**—J. Berdach [3] reports an **epidemic of cerebrospinal fever** which occurred in Trifail in 1898. There were 72 severe cases and from 26 to 30 abortive cases. Probably there were a number of other abortive cases which could not be positively diagnosed. It was a striking fact that the greatest number of cases as well as the greatest number of deaths were observed in persons between 20 and 25 years of age. The total mortality at this period of life was 26.6 % ; elderly persons to a considerable extent escaped the disease. The whole epidemic could be followed from the beginning to the end, and a very interesting chart is presented showing the portions of the settlement in which the different cases occurred. This demonstrates strikingly the fact that direct contagion had apparently nothing to do with the outbreak. The first case occurred a considerable distance from those next following, and the next outbreak was in a house still further away from either of the others. The same was repeatedly true throughout the epidemic. There was, on the other hand, no evidence of direct contagion. The houses in which the epidemic began were in most wretched hygienic condition, but a number of cases occurred in families of good social condition and living under good hygienic surroundings. The cases occurred both in the higher portions of the town and in the lower parts. Among some interesting observations which he details concerning the clinical course of the cases may be mentioned one case in which there was practically continuous vomiting, such as has been described by Leyden. Herpes appeared in practically all cases. It bore apparently no relation to the prognosis, but was an important diagnostic aid. Measles-like and scarlatina-like eruptions, and a roseola similar to that of typhoid fever, were repeatedly observed. Paresis of the facial nerve was seen in 85% of the cases, and was a very important symptom; it lasted only a few days. Hyperesthesia of the nerve-trunks, particularly of the ulnar, was very frequent. As an indication of the activity of the meningococcus intracellularis in causing the epidemic, he notes that besides the fact that this organism was found in the cases investigated bacteriologically, no case of pneumonia was known to have occurred in the region in which the epidemic was in progress; he thinks, therefore, that it can not be considered that the pneumococcus had any relation to this epidemic.

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[1] Deut. med. Woch., Dec. 14 and 21, 1899.
[2] Brit. Med. Jour., Nov. 18, 1899.
[3] Deut. Arch. f. klin. Med., Bd. LXV, Hefte 5 u. 6.

E. B. Doolittle[1] reports 10 cases of epidemic cerebrospinal meningitis, which occurred in a small village of about 800 inhabitants. The previous conditions of health or of hygiene seemed to have no influence upon the severity of the disease. The average duration in the nonfatal cases was 4 weeks; in the fatal cases, 7 days. In some cases the temperature reached 106° F. Seven of the cases were in children; these cases usually showed flexion at the thigh-, hip-, and knee-joints. In all cases he observed an eruption of herpes labialis. Albuminuria was found in every case. It is of interest to note that **10 cases of acute lobar pneumonia** occurred in the same district during the course of this epidemic of cerebrospinal meningitis.

J. W. Irwin[2] describes several epidemics of cerebrospinal meningitis in which he has had experience in Louisville. He has found **Kernig's sign** always present if looked for. He considers opium the most important of medicaments in the treatment, but in the later stages of the disease mercury and the iodids are likely to do good. He apparently noted the transmission of infection from one town to another through a woman who had just been engaged in nursing a case of the disease in the infected town and then went to the second town.

L. N. Boston[3] describes a case of cerebrospinal meningitis which had the unusual complication of **ulcerative endocarditis and abscess of the myocardium.** The meningococcus and the staphylococcus pyogenes aureus were both present, the meningococcus causing death when injected into a mouse.

## YELLOW FEVER.

**Etiology.**—E. Wasdin and H. D. Geddings,[4] in a report of their work as a commission to investigate the etiology of yellow fever, state that in 13 of 14 cases diagnosed as yellow fever they isolated the **bacillus icteroides,** and in the remaining case it was isolated by an independent observer. In 12 of 15 cases they obtained the organism from the blood during life when abstracted not earlier than the third day of the disease. In the 2 other cases the organism was obtained postmortem. [In cases which appeared to be other than yellow fever the bacillus icteroides was not obtained.] In 34 autopsies they obtained the organism in 30 instances. As a result of this and other work, they reach the conclusions that the bacillus icteroides is the cause of yellow fever; that it is naturally infectious, the degree varying with the species; that infection takes place by way of the respiratory tract, this local infection giving rise to the earlier manifestations of the disease. In the majority of the cases this is followed by secondary infection of the blood, and this stage may be complicated by infection of the blood with other organisms, or such infection may occur during a later stage of the disease. They believe that there is no evidence that the disease is primarily a septicemia, as there are certainly cases in which the bacillus can not be found in the blood. They believe that there is no causal relation between the bacillus "x" of Sternberg and yellow fever. So far as they have been able to discover, the bacillus icteroides has not been found in any bodies except those of patients dead of yellow fever. They consider the bacillus very susceptible to influences that are injurious to bacterial life, and its growth is readily controlled by disinfection. They think that there is reasonable possibility of the ultimate production of a serum for the treatment of the disease which will be more potent than the one which has been put forth by Sanarelli. They were able to determine that the domestic pig is incapable of infection by the bacillus icteroides when this organism is introduced into the digestive tract, and when fed to pigs, it does not produce the changes caused by the bacillus of hog cholera; hence they think that the two are different organisms.

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[1] Med. News, Aug. 5, 1899.

[2] Med. News, Sept. 2, 1899.

[3] Med. Rec., Sept. 2, 1899.

[4] Phila. Med. Jour., Aug. 19, 1899, p. 324.

G. Sanarelli,[1] in again discussing the **bacteriology of yellow fever,** replies to the critical statements that have been made concerning the bacillus discovered by him. The bacillus icteroides is unquestionably found in patients who are ill with yellow fever and in the bodies of those who have died of the disease, and recent investigations have shown its presence in a large proportion of cases, now practically amounting, in his belief, to every case. He believes that it is found only in those who are ill with this disease or in the bodies of those dead with the disease. While it does not produce characteristic yellow fever as in human beings, it does cause a peculiar fatty degeneration of the liver produced by no other organism, and Sanarelli considers that the symptoms and pathologic changes caused are those which are chiefly characteristic of yellow fever in human beings.

F. Vitale[2] first discusses the paper of Reed and Carroll concerning the identity of the **bacillus icteroides and the bacillus of hog cholera.** He disagrees with these authors concerning the identity of these two organisms, and notes the difference between their results and the results of others. He does not believe that the bacillus icteroides can produce infection through the digestive tract; and as an evidence of this, notes experiments upon rabbits, which animals are especially susceptible to the bacillus icteroides and to which he gave large amounts of cultures of these organisms without any effect. He thinks that the results of Reed and Carroll were probably due to symbiosis, the presence of the bacillus icteroides favoring the activity of the hog cholera bacillus and perhaps making the nonvirulent form virulent. Later,[1] in answer to Reed and Carroll's discussion of his work, he shows that in a number of observations upon dogs that died of various infections the bacillus icteroides produced more fatty degeneration, as a rule, than any other micro-organism, and states that Sanarelli made no claim that this organism alone produced fatty degeneration of the liver. As to the contention that the bacillus icteroides is the same as the hog cholera bacillus, he notes that the latter causes hemorrhages and abscesses besides the necrosis, and that the bacillus icteroides does not seem to produce these effects.

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[1] Med. News, Dec. 9, 1899.

[2] Med. News, Sept. 23, 1899.

A. Agramonte[2] believes that the **bacillus icteroides is not the cause of yellow fever,** and he thinks that proper methods of study will not show its presence with any frequency in the blood of persons who have died of yellow fever. He believes that it does not agglutinate with any regularity with the serum of yellow fever patients and that the organism causing the disease has not yet been found. In 37 cases he made cultures from the blood of the ear without in any instance obtaining the bacillus icteroides; in 31 cases he studied the blood from the veins with the same results; and in 23 autopsies he found the bacillus icteroides only 7 times, the bacillus "x" of Sternberg in 11 cases, the pyocyaneus in 7, and the colon bacillus very frequently. He found the bacillus icteroides in a case which he considers to be of undetermined character, possibly malarial, and in which the typhoid bacillus and malarial parasites were also discovered. The case, however, had been diagnosed by Wasdin and Geddings as yellow fever. He reports his results from injecting the heart blood from patients dead of yellow fever into guinea-pigs. No effect was observed, if accidental infections are excluded. He tried agglutination of the bacillus icteroides with the blood of yellow fever cases, and obtained it in only a few cases. He believes that agglutination occurs in those cases that show infection with the bacillus icteroides, though not regularly. Serum from convalescent yellow fever patients gave no protection to guinea-pigs inoculated with the bacillus icteroides. He used the **blood-serum of convalescents in the treatment** of yellow fever, and of 5 cases which he specially notes in his report, 4 recovered and convalesced rapidly, and in none of them was a hemorrhagic tendency shown. He thinks that the injections may prove of use. [It is impossible to reach a positive conclusion regarding the pathogenicity of the bacillus of Sanarelli, but there is a fair presumption in its favor.]

**Diagnosis.**—D. T. Laine,[3] after a collective investigation of **yellow fever** as it occurs **in the island of Cuba,** decides that there are no symptoms which are pathognomonic, but that the diagnosis must be made by the remission between the first and second periods and the occurrence of albuminuria, jaundice, and hemorrhage with the other usual symptoms. The mortality seems to be about 23%, but varies largely from time to time. Fiebre de borros is believed to be a mild form of yellow fever affecting the natives, while the so-called acclimatization fever is considered to be the same occurring in immigrants.

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[1] Med. News, Oct. 21, 1899.
[2] Med. News, Feb. 10 and 17, 1900.
[3] Med. News, July 1, 1899.

C. H. Tebault, Jr., [1] in speaking of the **diagnosis of calentura from yellow fever,** states that calentura differs as follows: The initial temperature is higher; the pulse becomes slow earlier in the disease; there is prolonged debility rather than coma or collapse; there is slight irritability of the stomach; jaundice is not common; the liver becomes increased in size; albumin is absent from the urine or present only in traces; there is no secondary infection; and while hemorrhage from the nose is common, other hemorrhages are rare. He thinks the lack of severe gastric symptoms and of albuminuria of great importance in the diagnosis.

H. A. Veazie [2] in discussing the diagnosis between **yellow fever and malaria,** notes that in yellow fever pains are chiefly situated in the knees and calves of the legs. The expression is excited, the eyes are watery, the tongue is covered with a white coat, the papillæ and edges are red, sore throat is common, inflammation of the parotid is frequently seen, and the liver is slightly enlarged, but the spleen is normal. The contrary of these facts is seen, as a rule, in malaria: the pains are general; the expression is dull; the eyes are congested but not watery; the tongue has a yellowish coat, is broad, and often indented on the sides; sore throat is rare, as is parotiditis; both the spleen and the liver are enlarged and tender.

**Treatment.**—C. B. Fitzpatrick [3] reports that by injecting horses with the filtrate of cultures of the bacillus icteroides he obtained after about 9 months **a serum** that would prolong the lives of guinea-pigs infected with the bacillus as compared with duration of life in control animals; and when the horse had been under treatment for more than a year, its serum, in doses of 10 cc. to 14 cc., was found to prevent death when guinea-pigs had been injected with 3 cc. of culture. A. H. Doty [4] describes a case of yellow fever which was treated with the serum prepared by Fitzpatrick. The man had a temperature of 104° F. with marked albuminuria, and was moderately ill. Three doses, 25 cc. each, of serum were given within about 8 hours. The fever rapidly declined and within a few days reached the normal. The serum was believed to have been useful in this case.

E. F. Nunez [5] reports favorable results from the use of **salol** and the **bitartrate of potassium** in yellow fever, giving these substances in order to produce, if possible, free excretion from the kidneys and antisepsis of the gastro-intestinal tract, because he believes that infection takes place from the gastro-intestinal tract or is increased from this source.

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[1] New Orl. M. and S. Jour., Sept., 1899.

[2] N. Y. Med. Jour., May 19, 1900.

[3] Med. Rec., July 1, 1899.

[4] Med. Rec., Aug. 26, 1900.

[5] Phila. Med. Jour., Nov. 11, 1899.

## PLAGUE.

**Etiology.**—F. G. Clemow [1] reviews the question of the **occurrence of plague in the lower animals** and the importance of such occurrence in infection. Monkeys have the disease, but apparently have little to do with spreading it. Rodents are found to have plague, particularly rats; but while it is uncertain to what extent rats act in spreading the disease, there is no question that they do have it, and sometimes aid in its propagation. Rats may become infected from the soil, from grain, from eating the flesh of other animals or of human beings dead of the disease, from infected rags, clothing, or dressings. It is highly probable that they are infected frequently by insects which infest them, particularly fleas. The manner in which rats become infected is uncertain. Bandicoots, mice, squirrels, guinea-pigs, porcupines, marmots, and rabbits certainly have the disease, but probably none of them have a very important part in its spread. Plague occurs only rarely in dogs, under natural conditions, and dogs practically play only an insignificant part, if any, in the spread of the disease; cats are but slightly susceptible, and probably do not spread the disease to any notable extent; horses are refractory under ordinary conditions; pigs are but slightly susceptible; sheep apparently do not spread the disease to any extent; goats are slightly more susceptible, but it is not known that they spread the disease; cows are scarcely susceptible at all; birds apparently have no part in the spread of the affection, but insects seem to have a great deal to do with it, though just how much is not yet positively determined.

M. F. Simon,[2] in a paper on plague in its relation to Singapore, decides that this disease is **not readily transmitted by sea** to places which are much nearer the tropics than those in which it occurs epidemically or endemically, and the spread of the disease will largely be overland, the bacillus gradually becoming accustomed to the hotter climates. If the disease does not occur in places where it is endemic, it is not readily communicated from one person to another. He considers that the danger of transmitting the disease from country to country over sea by cargo and baggage is much less than is commonly thought; and if a person acquires the disease before embarking, it is likely to abort, unless the infection has been very severe, when the patient travels by sea toward the tropical zone.

H. E. Deane,[3] from his experience with plague, considers **filth an extremely important etiologic factor.** The theory that the infection comes through abrasions of the feet he considers as not well supported by investigations. He has some doubt as to rats carrying the infection. There is more likelihood of indirect infection through clothing, etc., than of direct infection, since care will usually prevent the latter. Treatment by medicines is not particularly effective. Yersin's serum does not seem to be of much value. Deane has used the venom of poisonous snakes in 50 cases; and of these, only 22 died.

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[1] Brit. Med. Jour., May 12, 1900.

[2] Lancet, Jan. 20, 1900.

[3] Med. News, Feb. 24, 1900.

**Pathology.**—A. Weichselbaum, H. Albrecht, and A. Gohn[1] discuss the results from their study of the plague, during the course of which an epidemic outbreak occurred in Vienna. They recognize **two distinct forms**—bubonic and pulmonary. In the bubonic form there is a primary focus and the production of a bubo, and invasion of the near-by glands. In cases of general infection with buboes, the buboes arise in various parts of the body. In the form in which there is a primary bubo it is usually situated in the groin or in the axilla, sometimes in the neck. Sometimes the case progresses so rapidly that the primary bubo is not discovered; at times it may be in the tonsils or in the lymphatic tissues of the pharynx. The primary bubo is characteristic in that there is a marked necrosis of the gland with hemorrhage; one also finds hemorrhages in the mucous membranes and serous surfaces, as well as in the organs. The bacilli are found about these. The hemorrhages are produced by the action of the bacterial poison upon the walls of the blood-vessels. The spleen always enlarges. At times the disease may take the form of pyemia with metastatic foci scattered in various regions. The buboes may undergo resolution or may suppurate, in the latter case usually about the ninth day. The pulmonic form of the disease is relatively rare. It begins as a lobular pneumonia. In plague there may also be a metastatic pneumonia or an aspiration pneumonia, neither of which is the true primary pulmonary form. In some cases marasmus appears after the plague infection has passed off. The **points of entrance of infection** are chiefly the skin, the mucous membranes of the mouth, nose, pharynx, trachea, and bronchus, and the parenchyma of the lungs, and there is even observed infection through the stomach or intestines. Only a slight destruction of tissue is necessary to give rise to infection; mere shaving of the skin of animals is enough to give opportunity for introduction of the germs. Diagnosis may be made by examining the sputum in the pneumonic form or by incising or puncturing a bubo. In the bubonic form occasionally the bacilli are found in the blood, in the saliva, or in the urine. A positive agglutination test is valuable in diagnosis, but not infrequently the test may be negative when plague exists.

**Symptomatology.**—J. Cantlie,[2] in a general discussion of plague, speaks of the **rat and the mouse as the most important factors in the spread** of the disease. The incubation is from 3 to 5 days, and if a voyage of 5 days has been undertaken, the persons on board ship may be considered free from danger. There is no danger of infection of others unless the ship is infested with rats. The bacilli die after about 6 days in grain, clothing, etc., but there are always rats about the grain, and therefore grain is likely to be infective for a long time. He describes the general characteristics of the disease. He states that albumin is not common in the urine in a first epidemic; in recurrences it is found in most of the cases. The most fatal period is the third or fourth day. The death-rate among Chinese and native Indians is about 90%, this being lessened when they are treated in European hospitals. About 50% of Europeans die. The prognosis can scarcely ever be stated. Cantlie insists upon the importance of destroying rats if there is danger of a plague epidemic. Persons who have been exposed should be detained in special camps for at least 7 days, and if they are ill, should be injected with Haffkine's prophylactic. After inoculation they may be considered exempt from isolation for 6 months. The cargoes of ships should receive careful attention. Grain should be thrown overboard; wheat should be allowed to float about for a week or two in the sea-water; sugar should be cast away; cotton, silk, and wool should be placed in isolated sheds; and all the cargoes should be carefully examined for rats, and if any are found, the portions of the cargo containing them should be destroyed.

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[1] Wien. klin. Woch., Dec. 14, 1899.

[2] Practitioner, Nov., 1899.

W. J. Simpson,[1] in a general discussion of plague, notes the importance of observing in the early part of epidemics the **slow and irregular way of advance** of the disease in the community and the lack of typical characteristics in the early cases. The most dangerous forms to public health are the obscure pneumonic and the ambulant varieties. The pneumonic form is characterized often only by cough and fever and exceedingly severe prostration. It is particularly dangerous because of the large numbers of bacilli given out in the sputum, and can be diagnosed certainly only by finding the bacilli in the sputum. The ambulant form is also dangerous, because these patients come in contact with others constantly. Rarely the bacillus may be found in the blood, but usually one can diagnose the cases only by developing a very careful history and by eliminating other factors. In the septic variety the bacilli are often found in the blood in large numbers. This form is usually recognized. It comes on with extreme rapidity and causes death in a short time. The chief pathologic conditions found are congestion and hemorrhage with parenchymatous degeneration of the liver and kidneys. The treatment of plague is purely symptomatic, but the point insisted upon is that there should be careful disinfection of all secretions, and, particularly, infectious matter that has not been disinfected should not be cast into the drains, since there is great danger of infecting rats and thus propagating the disease. In a later article Simpson [2] discusses the plague from several aspects, and directs particular attention to the mode of outbreak at Asuncion, because of the **danger to** the remainder of the **American continent,** since plague has acquired a locality for diffusion from this point to other parts of America. Concerning diagnosis he states that when a patient is seen with fever and with the peculiar physiognomy of plague, and with hesitating speech, staggering gait, and coated tongue, one should always make examination for buboes or disease of the lung, and the blood, sputum, and buboes should be examined for bacilli. The obscure cases are the most dangerous in the spread of the disease from country to country.

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[1] Brit. Med. Jour., Sept. 16, 1899.

[2] Lancet, April 14, 1900.

W. C. Hossack [1] reports 5 cases of **a form of insidious plague pneumonia** which he considers extremely dangerous in the dissemination of the disease. The symptoms came on very gradually, the local signs were slight, and there were none of the usual characteristic signs of plague, such as disturbance of the speech and the peculiar tongue. An important feature was the rapidity and feebleness of the pulse, which was noticed from the early stages of the affection.

W. H. Kellog [2] reports that his bacteriologic investigations of the case of a Chinaman who died in **San Francisco** showed the presence of the plague bacillus. The man had been treated merely for a bubo, which was thought to be gonorrheal. He died, however, with vomiting, profuse diarrhea, and collapse. Kellog has also made postmortem examination of 3 other Chinese, and 2 of these he thinks were plague cases, but he was unable to prove this because the bodies were in an advanced stage of decomposition. The Health Board work in the Chinese quarter in San Francisco seemed rapidly to stamp out the disease before any really deep hold had been gained.

**Treatment.**—W. M. Haffkine [3] presents his report on **preventive inoculations** for plague. He demonstrates the fact that large communities may be inoculated within a short time. At Hubli, for instance, practically all of **50,000 inhabitants were rapidly inoculated.** The difference in mortality in those inoculated and those uninoculated averaged from 80% to 90%. Instances which he quotes were results at Byculla, where 12 cases and 6 deaths occurred among 172 uninoculated inmates of the jail, while but 2 cases, with no deaths, occurred among inoculated inmates. Very similar results were seen at Umerkadi jail, where 127 uninoculated inmates furnished 10 cases and 6 deaths, while 147 inoculated gave no deaths and only 3 cases. At Undhera 27 cases, of which 26 were fatal, occurred in 64 uninoculated persons; 71 who were inoculated furnished only 8 cases with but 3 deaths. Similarly favorable results seen in a number of other places are described. Haffkine believes that it is demonstrated that the prophylactic has the same effect in man as it has in laboratory animals. It apparently had no effect upon cases in which the plague was incubating at the time of the inoculation. The length of time through which the immunity lasted was not well determined. The dose was 2.5 cc.

V. Tchistowitch,[4] in discussing the epidemic of plague that occurred in the Russian village of Kolobovka, states that there were, in all, 24 cases of plague, of which 23 were fatal. He describes a series of the cases and the cultures obtained therefrom. The last patient stricken recovered. Shortly afterward preventive inoculations with **Haffkine's prophylactic** were begun, and after great difficulty almost 4000 people were inoculated, only 40 people in the neighborhood remaining uninoculated. After this no more cases of plague occurred. Together with the use of the prophylactic, quarantine regulations were strictly enforced.

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[1] Brit. Med. Jour., Feb. 1, 1900.

[2] Jour. Am. Med. Assoc., May 19, 1900.

[3] Proc. Roy. Soc., vol. LXV, No. 418.

[4] Ann. de la inst. Pasteur, Mar., 1900.

S. Mallannah,[1] in investigating Haffkine's plague prophylactic, found that there was an immunizing substance giving proteid reactions in the sediment after filtration; the clear filtered fluid also gave proteid reactions and was protective in much smaller doses. The **immunizing substance was isolated** by Brieger's method; it then gave no proteid reaction. It was a gray amorphous substance, soluble in water. There is a proteid substance which produces pus which is elaborated by the plague bacilli. This seems to be found in the bodies of the bacilli, and suppuration or induration does not occur when the extracellular immunizing substance or the filtrate of the prophylactic fluid is used.

C. B. Stewart [2] has found that plague bacillus cultures when given in small doses are fatal, while increased doses are less fatal. He therefore decided that there was some substance in the cultures which produces immunity, and he thought that this is probably in the filtrate, as the cultures were young and living. He found that the filtrate alone possesses the power of modifying the attack, while a suspension of sterilized microbes has bactericidal properties. He therefore considers that the **deposit** of the cultures **possesses bactericidal properties**, while the **fluid portion possesses the property of conferring immunity.**

A. Calmette [3] reports the results which he obtained in working with Salimbeni in the epidemic of plague at Oporto. They used the **serum of Yersin.** While before the serum was employed the hospital mortality was 33%, after the use of the serum on 104 cases the mortality dropped to 13%. Of 9 cases that died, they present the following notes: 3 died within 16 hours after entrance to the hospital, 1 after 24 hours, 1 had a plague peritonitis, 1 had a plague meningitis, another had tuberculous meningitis together with the plague, and the ninth case occurred in a woman who had recently given birth to a child, and who had with the plague a puerperal streptococcic infection. They insist that it is necessary to inject large quantities of the serum, and to reinject each day until the temperature indicates that there is no longer danger of reinfection. As a demonstration of the effect of the serum upon the disease they note that in a case in which before injection they obtained from culture of one drop of blood 32 colonies of bacilli, after the first injection they obtained only 1 or 2 colonies from the same quantity of blood; and when the second injection was given and a culture taken shortly afterward, the blood was entirely sterile, the bacilli having completely disappeared. **Injections should be repeated** until convalescence is established, since deposits of bacilli are likely to be present so long as there are still any signs of disease. The injections should be undertaken daily, and should consist of from 20 cc. to 40 cc. of serum. They injected as much as 320 cc. within 6 days in one woman, and although treatment was begun only on the fourth day, and the patient had an exceedingly severe plague septicemia with multiple buboes, she recovered completely. In plague pneumonia they did not hesitate to introduce the serum directly into the veins in one dose of 20 cc., and they state that they did not lose a single case of plague pneumonia treated in this way. They consider the use of the serum even more valuable when used for prophylactic purposes in preventing the spread of plague. They did not see a case of plague in persons who had received a prophylactic injection. The duration of immunity is only from 20 to 25 days, however. They believe that the coincident use of Haffkine's vaccine and Yersin's serum is useful, but they think that the effects of Haffkine's vaccine are unpleasant, and that, as a general rule, it is not so useful as the serum.

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[1] Brit. Med. Jour., May 12, 1900.

[2] Brit. Med. Jour., Mar. 3, 1900.

[3] Gaz. hebdom. de méd. et de chir., Nov. 5, 1899.

The special correspondent to the Lancet in India writes [1] that the official reports concerning the use of **Lustig's serum** show that there are no indications that the remedy will prove efficacious on further trial. The case mortality under this treatment was 69.7%, which was not better than the mortality in unselected cases treated by ordinary means. The great error in the use of the serum is in the fact that the serum is given to selected cases. It was not administered in moribund cases or in cases on the first day of their admission to the hospital. As to the latter point, it is well recognized that of these cases about 30% died within 24 hours of admission to the hospital; hence such severe cases were excluded from the statistics, because no attempt was made to use the serum in them. There was also no evidence that early use of the serum lessened the mortality correspondingly. A. Lustig and G. Galliotti [2] consider that their method of inoculation against plague has been proved to be effective and innocuous. They believe that culture liquids owe their immunizing power to the nucleoproteid which they contain. It is much more advantageous to use this than a liquid from the culture, because other substances which are harmful are often injected in this way. There is no necessity for heating, and therefore no immunizing power is lost, and the dried nucleoproteid may be used and administered in definite doses.

J. M. Atkinson [3] describes a case of plague which occurred in a Scotchman 30 years of age, and was moderately severe. His treatment was 2 grains of **carbolic acid** every 2 hours, over 200 grains of the drug being given within 3 days. The temperature was considerably lowered, the vomiting was relieved, the mental depression became less, and there were no symptoms of poisoning. It was thought that the carbolic acid produced a cure.

H. E. Deane,[4] in continuing his article on plague, describes 19 cases in which he used **snake-venom** with only 6 deaths. He thinks that the treatment promises successful results.

## ERYSIPELAS.

G. E. Pfahler [5] reports his **bacteriologic investigation** of 8 cases of erysipelas, and describes **diplococci** which he found in the whole series of cases and which he believes are the cause of erysipelas or of a disease which is clinically identical with it. The organism was found in pure culture, and it produced a disease entirely similar to erysipelas in rabbits in the 4 instances in which it was inoculated; the same organism was also recovered from the tissues of the inoculated animals. The diplococcus was about the size of the pneumococcus. It nearly always occurred in pairs, frequently singly, more rarely in chains. It is encapsulated and it stains well with gentian-violet and carbol-fuchsin, but poorly with methylene-blue solution. It is nonmotile, aerobic, and grows best at 37° C. It clouds bouillon and forms opaque, white, well-defined colonies within 24 hours on agar. Larger colonies form on blood-serum. It does not liquefy gelatin, and it does not alter the reaction of milk. The organism was obtained by cleansing the skin and making preparations of the pus from pustules; or by passing a needle through the skin, then pressing blood from the puncture, and making cultures from this.

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[1] Dec. 16, 1899.

[2] Brit. Med. Jour., Feb. 1, 1900.

[3] Lancet, Dec. 9, 1899.

[4] Med. News, Mar. 3, 1900.

[5] Phila. Med. Jour., Jan. 3, 1900.

Chantemesse and Ray [1] have found that a **polymorphonuclear leukocytosis,** parallel with the severity of infection, occurs in erysipelas. This was not observed unless they warmed the finger of the patient regularly before taking the blood count. The number of leukocytes found when the blood is taken from the cold finger is usually only in the neighborhood of one-half the number found when the blood comes from the warmed finger. This is due, they state, to the fact that the leukocytes lie along the walls of dilated small vessels in the slowest portion of the current. Contraction of the vessels causes the blood flow to become more rapid, and hence the stagnant leukocytes are driven on into the larger vessels and their number in the peripheral circulation seems to be lessened.

Prat [2] describes a case of **erysipelas** of the face **in a Senegal negro** 18 years of age. It has been considered by some that the negro is entirely refractory to the streptococcus, but this case and others observed demonstrate that this is not true. The evolution of disease in this case was quite the same as that often seen in white subjects, the disease beginning with sore throat and enlargement of the cervical glands.

## RELAPSING FEVER.

G. O. Ward [3] describes a case which he believes was relapsing fever which occurred in an Armenian immigrant who had recently come from Asiatic Turkey. He was admitted with a temperature of 104° F., but with no evidences of typhoid fever, and malarial parasites could not be found. The temperature remained high for a time, when it rapidly fell to subnormal. Eight days later it rose very rapidly from 97.8° F. to 105.5° F., and **leukocytosis** occurred at the same time. It fell again 3 days later, and convalescence soon became established. The spirillum was not looked for until the occurrence of the relapse, and was not found then.

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[1] Presse méd., July 1, 1899.

[2] Gaz. hebdom. de méd. et de chir., Nov. 9, 1899.

[3] Boston M. and S. Jour., Jan. 11, 1900.

## WEIL'S DISEASE.

W. Richter [1] reports a case of Weil's disease which began with the appearance of typhoid fever except that there was an eruption of herpes and the temperature rose unduly rapidly. There was no splenic tumor, however, and no eruption, and soon the liver enlarged, the urine showed bile, and severe muscular pains came on. The patient became jaundiced and very stupid, with anuria, and died suddenly. Necropsy showed hemorrhagic nephritis with hyperemia of the mucous membrane in general and edema of the lungs; the liver was not enlarged, was somewhat fatty, and was yellowish-brown in color.

## MALTA FEVER.

W. Cox [2] records a case of **Malta fever** which occurred **in Porto Rico.** The patient was a man of 30 whose case had been diagnosed as remittent malarial fever. He had been confined in an extremely damp and foul guard-house, where fever began on January 11th. It was associated with general pains, and chills occurred at first. The daily remissions were very pronounced, the temperature reaching normal nearly every day, but rising to 101° F. or 102° F. Beginning February 27th, there were several days of apyrexia; then fever began again, and continued until March 27th, when he was again free from fever until April 7th. He was discharged from the hospital 10 days after this. There were 91 days of fever altogether, with marked daily remissions and a general undulatory character of the fever curve. The blood reacted to a culture of the Micrococcus melitensis in a dilution of 1 : 50.

A. Brunner [3] describes a case of Malta fever in which there was a positive **agglutination test,** and **puncture of the spleen** showed a micrococcus which had the characteristics of the Micrococcus melitensis. The latter organism was agglutinated by the patient's blood-serum in high dilution.

## VACCINIA.

Hervieux,[4] in discussing the **causes of the reduction in the virulence of vaccine** in hot countries, and the methods of remedying this, states that it has been definitely shown that the attenuation of the vaccine is caused by the high temperature—a fact which has been recognized for a long time by military surgeons in the French colonies. He gives a series of instances to demonstrate the fact that when vaccine is kept cold and used without being exposed to a high temperature for any length of time it remains quite as satisfactory in the tropics as elsewhere, while after exposure to heat it rapidly loses its activity. He thinks that there is no doubt that heat is the chief factor in reducing the virulence of the virus, and moist heat is more harmful than dry heat. The influence of winds may sometimes be seen, the sirocco, in Tunis, being distinctly harmful. There are also other factors which influence the virus, particularly keeping it for a long time. He believes that glycerinated vaccine which is introduced into a tube and the tube sealed by heat should be at once placed in an ice-chest in order to insure the preservation of its activity, and he thinks that in carrying vaccine about in hot countries one should either carry it in an ice-chest or wrapped in cloths which are kept constantly wet and are placed in a current of air in order to reduce the temperature through evaporation. He insists that while smallpox is a constant menace to health and life, vaccine is useful in protecting from smallpox through only about 7 months in the year in any countries that are subject to great heat.

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[1] Deut. med. Woch., Oct. 26, 1899.

[2] Phila. Med. Jour., Sept. 9, 1899.

[3] Wien. klin. Woch., Feb. 15, 1900.

[4] Bull. Acad. de méd., Feb. 13, 1900.

D. P. Austin,[1] in discussing **vaccination,** insists that it **must be carried out repeatedly** until no result is obtained in order to be assured that immunization has been secured. He believes that the single use of the virus is of relatively little value, as immunity may not be secured by one vaccination. He has found repeatedly that several vaccinations may take, but that afterward no result is obtained, and he considers security attained only when an attempted vaccination fails.

W. E. Fowler[2] describes a case of **general eruption after vaccination.** This occurred in a negro of 30 who was a subject of tertiary syphilis. Three weeks after vaccination a number of large vesicles appeared about the point of vaccination and a papular eruption was seen on the face and neck and soon spread over the body. The vesicles became papular and had the odor usually noticed in smallpox. It is thought that syphilis was possibly a factor in the condition.

F. G. Atwood,[3] in discussing **bad results** from vaccination, states that most of these are **due to imperfectly prepared virus** and to carelessness in carrying out vaccination. He has found in many cases that the subcutaneous injection of glycerinated lymph gives good results.

W. G. Mortimer[4] reports that **after revaccinating** a man of 23 large **warty disfigurement** of the fingers and chin **rapidly disappeared,** and vanished entirely within a week. Numerous other treatments had been used on the warts, but had had no effect, and this result was entirely unexpected.

## VARIOLA.

W. M. Welch[5] reports upon the character of the cases of smallpox which he has recently seen in the Municipal Hospital in Philadelphia. He has **observed frequent errors in diagnosis** among practitioners, and this he attributes to imperfect instruction and to the mildness of the cases which have been recently seen. He strongly recommends opening hospitals for infectious diseases for the instruction of students. The recent cases have been of the mildest type that Welch has ever seen.

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[1] Medicine, Feb., 1900.

[2] Med. Rec., Sept. 23, 1899.

[3] N. Y. Med. Jour., Dec. 2, 1899.

[4] Brit. Med. Jour., April 7, 1900.

[5] Phila. Med. Jour., Nov. 18, 1899.

In many cases it was impossible to count as many as a dozen pustules, even when the patient had never been vaccinated. Except that it was milder, the onset of the disease was much the same as it is in severe forms. After the eruption came out, many patients seemed entirely well. Within one year he has seen 128 cases with no deaths, and only 2 or 3 cases were at all serious. One hundred and ten of these were vaccinated, 17 were vaccinated in infancy, and 1 after exposure to the infection; 122 of the patients were colored. Of the 17 patients who had been vaccinated in infancy all but 1 were over 15 years of age, so that there had been sufficient opportunity for the disappearance of the protective power of vaccination. The eruption in many cases ran an abortive course; oftentimes the papules became solid conical elevations with a small vesicle at the summit. Desiccation was rapid. The lesions seemed to involve only the outer epidermis and the layer of the cells immediately over the papillæ. There was little or no secondary fever. The difficulties in diagnosis were chiefly between varicella, impetigo contagiosa, and pustular syphiloderm. Concerning varicella, Welch states that the lesions are distinct vesicles containing clear serum, and that they appear first on the parts of the body covered with clothing. They come out in groups and vary greatly in size; they are unilocular; their covering is so delicate as to be easily broken; they feel soft and velvety; they rarely look umbilicated, and then only when desiccation begins in the center; they run their course in from 2 to 4 days; the crusts are brown and friable, and when they fall, leave red spots instead of the pigmented spots that follow smallpox. Scars are rare. In impetigo contagiosa there is no initial stage; it does not begin as papules, but as vesicopustules, which appear on the normal skin and are superficial, and enlarge by peripheral extension, often attaining the size of a 10-cent piece and having a flat appearance. The crusts are friable and only lightly adherent, and crumble off in small pieces. Scars do not result. Also the patient often infects himself by scratching, one area being infected after another. In syphilis it is to be remembered that the constitutional symptoms are milder in the initial stage. The papules are not shot-like. In syphilis the vesicle forms at the summit of the papule and becomes turbid and then pustular, and is situated upon an indurated base. The lesions appear in crops, they are not umbilicated, but some of them tend to ulcerate; the scabs are brown and friable, and after falling leave a coppery hue.

R. W. Woodson [1] describes the epidemic of smallpox which he saw at Holguin, and draws **conclusions from 1200 cases.** Probably at least 1000 more cases had occurred, but escaped to the mountains. In many regions the epidemic was extremely virulent, probably because vaccination had been but little practised. The inhabitants had become severely depleted physically during the Spanish dominion, malarial cachexia had reduced their health in almost all instances and made them susceptible, and the natives are always very crowded and under extremely bad hygienic surroundings. The epidemic occurred during the rainy season and hot months. This is explained by the fact that the people are most overcrowded at that time. Woodson gives a description of the methods used to isolate the patients and manage them. The general mortality was 9.6%; the mortality in the period from May 1, 1898, to November 1, 1898, was 29.5%. Males and females showed almost the same mortality, though more females were affected. The mortality rate in those that had been vaccinated was *nil.* The greatest susceptibility was among children of from 5 to 14, with a low mortality rate, while in children under 5 years the mortality was almost double. The susceptibility of female infants seemed to be greater than that of males. Of 505 discrete cases, only 9 were fatal. In the confluent cases the mortality was 13.83%, while of the malignant cases 70% died. The malignant cases were more fatal in women than in men, contrary to the usual experience. The youngest patient was 3 days old; the oldest, 80 years; both died. The patients were treated with baths of 1 : 2000 bichlorid and by being placed in the open air. Cosmetic treatment was impossible in a large number of cases.

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[1] Phila. Med. Jour., Nov. 18, 1899.

C. O. Probst [1] reports upon an **epidemic of smallpox** which was observed in numerous regions in Ohio in 1898. The first case appeared on April 6th, and during the following 14 months 1882 cases with 30 deaths were reported, 45 of 88 counties being infected and the disease being observed in 61 cities and villages, besides invading the rural districts. The form of the disease was usually mild, and it was often unrecognized. The preliminary fever was often slight or absent, and secondary fever was seen only in a small number of cases. There were, however, a few cases of the hemorrhagic form. Vaccination seemed to be completely protective even when done many years before, and the question as to whether the patient had been vaccinated or not became an important one in the diagnosis of obscure cases. An inquiry from house to house, with careful history of the families, showed that the disease had begun by attacking the unvaccinated members of the families. **Six outbreaks** were **traced to a traveling theatrical company.**

J. F. Jepson [2] discusses the epidemic of smallpox which occurred at Wheeling, W. Va., in 1895, in which there were 146 cases. Sixteen deaths occurred, all of them in persons who had not been vaccinated. The mortality among those who had not been vaccinated was 26%, while in those who had been vaccinated there was no serious case, and in those recently vaccinated no case was seen. Second attacks occurred in a number of persons, and 3 of the worst cases were second attacks of smallpox; Jepson believes that recent vaccination is a better protection than previous attacks of the disease. He discusses the control of the disease and insists upon the necessity of vaccination.

**Treatment.**—N. J. Kowtowtschicoff [3] recommends **repeated vaccination** as a treatment for smallpox **even in the suppurative stage.** This should be undertaken twice daily, and the inoculation should be carried out each time in several areas. In 3 cases thus treated he believes that he was able to make the suppurative stage of the disease much less severe than it would otherwise have been. He thinks that the treatment should be instituted in the prodromal stage; but if the case has not been seen sufficiently early for this, then on the first or second day of the invasion, or, if necessary, even later.

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[1] Jour. Am. Med. Assoc., Dec. 23, 1899.

[2] Ibid., Dec. 23, 1899.

[3] Zeit. f. klin. Med., Bd. XXXVIII, Hefte 1, 2, u. 3.

J. Biernacki and N. Jones[1] describe 8 cases of smallpox that were treated by daily doses of 60 grains of **salol.** They believe that by using this drug general pustulation may to some extent be prevented; in one case, after the maturation of the pustules had begun, the salol seemed practically to abort their course. The irritation of the skin is much reduced by salol and the course of the case is much more rapid under its influence. Scarring is diminished and there is said to be less odor.

C. Begg[2] describes valuable results from the use of salol in smallpox, stating that it relieves the irritation of the skin and the desire to scratch, and that the vesicles do not frequently suppurate. In one case which was confluent suppuration occurred in only 2 vesicles. He gave the drug in doses of 15 grains every 4 hours.

H. A. Ingalls[3] reports that in conjunction with Yeager he has treated 36 cases of variola with **baths of bichlorid** without any deaths. In this number there were 13 confluent cases and 1 hemorrhagic. He thinks that the suppurative fever can be shortened from 4 to 6 days, and that the mortality may be reduced to a minimum. Pitting is far less and the indescribable odor is entirely absent. The period of desquamation is also greatly shortened.

## GONORRHEAL SEPTICEMIA.

P. Cardile[4] reports the case of a woman who 1 month after the onset of an attack of gonorrhea showed the signs of a **pleural exudate.** In the fluid obtained by aspiration Cardile found cocci which, both morphologically and upon cultivation, showed the characteristics of gonococci. The fluid was aspirated 3 times. After this, complete recovery ensued.

Potain[5] describes a case in which there was an association of acute gonorrhea in a woman with **multiple arthritis** and a **systolic heart murmur.** There were no other signs of actual disease of the heart except some loss of clearness of the heart sound, yet Potain does not hesitate to pronounce the case one of endocarditis due to gonorrhea. The area of aortic dullness was also enlarged. This he quite as boldly attributes to gonorrheal aortitis. [A large number of cases of cardiac disease in association with gonorrhea might be collected from literature, but instances of undoubted gonorrheal endocarditis or myocarditis are rare.]

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[1] Brit. Med. Jour., June 2, 1900.

[2] Brit. Med. Jour., Jan. 6, 1900.

[3] Jour. Am. Med. Assoc., April 28, 1900.

[4] Clinic. med. ital., No. 9, 1899.

[5] Bull. méd., Dec. 13, 1899.

F. Duplant and M. Pehu [1] report 2 cases of **gonorrheal arthritis of the hip-joint,** noting the rarity of the involvement of this joint. The affection is often obscure, there being little redness and evidence of inflammation, and it often resembles sacro-iliac disease and sciatica. It is differentiated from these conditions chiefly by the situation of the tenderness and of the pain upon movement of the joints. In the cases of arthritis there was severe pain in the inguinal region and upon palpation in Scarpa's triangle; sudden shock against the great trochanter caused no special pain. The tenderness was very superficial, and the authors believe that this suggested that the disease involved chiefly the periarticular tissues. One of the cases whose arthritis came on about 2 months after infection was cured entirely after about 2 weeks' treatment. In the other case the disease had lasted a year at the time of the report, and there was little change in the man's condition, the limb being almost useless and the man still suffering intensely. These cases show the variable course the disease is likely to pursue.

R. Pelisse [2] states that **blennorrhagic rheumatism** frequently occurs, and that the occurrence of one attack does not protect from subsequent development of gonorrheal rheumatism; the joint condition, indeed, frequently appears within a few days after the onset of a second attack of gonorrhea, and at times one may observe recurrence of the joint symptoms after each new infection. If repeated recurrences are seen, the arthritis is likely to change from the pseudophlegmonous form to the appearance of progressive deforming arthritis of the nodular variety.

Eichhorst [3] describes a case of gonnorheal **sclerosing inflammation of the muscles.** This is a rare complication of gonorrhea, usually observed in men, and generally affecting the muscles of the lower extremities. The case presented is still rarer, because the extremity involved was the right arm. A mass developed in the right side of the upper arm, and there was a gradual formation of a spindle-shaped collection in the tensor fasciæ muscle, which was first accompanied by pain. The pain afterward disappeared, but the mass remained.

## PNEUMOCOCCUS INFECTION.

Rendu [4] describes his observation of a **pneumococcic arthritis** which occurred in a man of 66 who had a frank attack of acute pneumonia; 3 weeks later the temperature was still somewhat elevated and the auscultatory signs still persisted markedly. There was then a sudden increase in the temperature and the appearance of an acute arthritis of the left sternoclavicular articulation and of the knee. The latter joint did not suppurate, and recovery followed upon the aspiration of 120 cc. of fluid. The other joint suppurated. The fluid from both joints contained large numbers of pneumococci.

W. Osler,[5] in discussing the diagnosis between **pneumococcic**

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[1] Rev. de méd., Nov. 10, 1899.

[2] Thèse de Paris, 1899.

[3] Deut. med. Woch., No. 42, 1899.

[4] Gaz. des Hôp., June 1, 1900.

[5] Phila. Med. Jour., July 1, 1899.

**meningitis** and cerebrospinal fever, divides the pneumococcus form into that which is a complication of lobar pneumonia, that resulting from local infection, and primary pneumococcic meningitis, which is an evidence of general infection by the pneumococcus. Those symptoms of the form complicating pneumonia which help to distinguish it from cerebrospinal fever are the severe headache and early delirium, the rarity of spinal symptoms, the results of lumbar puncture, and, most important clinically, the almost universally fatal result in pneumococcus meningitis. The pneumococcic cases that result from local infection have, in his experience, been 3 in number, 2 of them having followed surgical procedures. Two cases of primary pneumococcic meningitis are recorded.

Cohen[1] reports a case of **pneumococcus meningitis of obscure origin** which occurred in a woman of 35 after abortion. Lumbar puncture showed the presence of the diplococcus pneumoniæ, and at autopsy ulcerative endocarditis and purulent meningitis were found, and there was a purulent endometritis with retention of a portion of the placenta. Cultures from the spleen, the heart, and the endometrium showed the pneumococcus.

## PYOCYANEUS BACILLEMIA.

N. E. Brill and E. Libman[2] report a case of **pyocyaneus bacillemia** which occurred in a man of 23 who was admitted with jaundice, irregular fever, and prostration. The staphylococcus aureus was found in a blood culture. The man improved greatly within 2 weeks and was out of bed. At that time his red cells were reduced to 900,000 and the hemoglobin to 20% and his general appearance was that of pernicious anemia. He developed a metastatic abscess, however, the temperature rose, the jaundice and prostration returned, and the patient died with severe nervous symptoms. Cultures from the blood during life and from various organs after death, as well as sections from the organs, showed large numbers of the bacillus pyocyaneus; the metastatic abscess contained the staphylococcus. There was a peculiar pigmentation of the skin in small patches, which the authors think was due to degenerative atrophy of the adrenal bodies resulting from the toxemia. They divide cases of pyocyaneus infection into those in which the bacillus was found during life or postmortem in cavities communicating with the air, those in which it was found during life in cavities or exudates not communicating with the open air, and those in which there was a general bacillemia.

## AEROGENES CAPSULATUS BACILLEMIA.

N. B. Gwyn[3] **isolated** Bacillus aerogenes capsulatus **repeatedly from the circulating blood** of a young woman of 18. The patient had chorea insaniens, and ultimately died. Shortly before death streptococci were obtained from the blood, there being probably a terminal infection. No autopsy was made. This is the first record of the isolation of this organism from the blood during life. There were no signs of emphysema in this case, and apparently the bacillus may exist in the blood without producing gas.

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[1] Münch. med. Woch., Nov. 21, 1899.

[2] Am. Jour. Med. Sci., Aug., 1899.

[3] Johns Hopkins Hosp. Bull., July, 1899.

## BERIBERI.

F. Clark [1] reports an epidemic of beriberi in a foundling house. Sixty-nine children were isolated as suffering from the disease. All these children were between 4 and 7 years of age; all slept in adjoining rooms on the ground floor. The hygienic conditions were very good, the dietary was generous, and there was no overcrowding. After the removal of those affected, the 27 remaining children showed no symptoms of the disease. There had been beriberi in a neighboring institution, the inmates of which came in contact with the children in the foundling house, but no direct source of infection could be traced. The first cases in the foundling house occurred in children who required surgical dressings; they were attacked only 2 days before the others, however, and probably were especially susceptible owing to their depressed condition rather than to the existence of surgical conditions.

## INFECTIONS OF OBSCURE ORIGIN.

W. Gabel [2] describes a peculiar disease, probably infectious, which is apparently due largely to the climate. It occurred around 3 towns in the southern part of Herzegovina, all of which are situated in high valleys surrounded by mountains. In all these places the climatic conditions are practically subtropical, there being a rainy spring followed by a hot, dry summer, with very trying nights. The disease is most frequent in July. There are usually no prodromal symptoms of any consequence. The disease begins with a general feeling of distress and headache, but no chill; there is often disturbance of the gastro-intestinal tract. The temperature rises rapidly, and a striking early symptom is muscular pain, particularly in the lower extremities; there are often convulsive movements of the lower extremities. The fever lasts 4 or 5 days and ends by crisis. The Widal reaction was negative; Gabel considers, however, that it is quite possible that in some of the cases typhoid may have been present, as he finds that a very acute onset in typhoid fever is not uncommon in the region in which these cases occurred. One striking characteristic of the disease, however, is that there is frequently a relapse. In this way it greatly resembles relapsing fever. Bacteriologic and direct microscopic examinations of the blood were always negative.

## LEPRA.

D. W. Montgomery,[3] in discussing **contagion in leprosy** as observed in San Francisco, refers to a case which he has previously mentioned in a man who had never been out of the United States, but who had mixed a great deal with Chinese and had acquired leprosy. He states that he has also seen a number of instances of alien-born persons who had acquired leprosy after a long residence in the United States. He had also seen 5 Chinese who had been in America long enough before the development of the disease to make it probable that the disease had been acquired in this country.

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[1] Brit. Med. Jour., May 12, 1900.

[2] Wien. med. Woch., 1899.

[3] Canad. Pract. and Rev., Mar., 1900.

J. R. MacMahon [1] records **a case of leprosy** in a man who had always lived in Great Britain. His chief point in the report is that he had seen a patient with leprosy who had stayed at a hotel where the bedding was not regularly changed when different persons occupied the rooms, and he suggested that this may be one of the ways in which the disease is propagated.

J. D. Gimlette [2] reports a case of leprosy which occurred in a clerk and interpreter in the police court at Pekin. The case had been diagnosed at first as rheumatic inflammation of the tendon sheaths, and afterward as beriberi, but subsequently the man developed typical mixed leprosy. It was evident at once that there was considerable **opportunity for infection** of others because of the man's employment. Gimlette, however, does not believe that this danger was really great, since the condition was recognized soon after the developement of sores, and he does not think that a patient is a source of danger until open sores have developed.

J. A. Silberman [3] describes the case of a man of 52 who had previously had syphilis, and who was confined during the Cuban rebellion in a foul dungeon for 80 hours. When seen he was very anemic and had albuminuria, and there was a leukoderma of the legs, with hyperesthesia of the soles of the feet. There was no swelling or anesthesia, but the symptoms **resembled leprosy.** It was considered to be either syphilitic leukoderma or dungeon scurvy.

**Treatment.**—J. Carrasquilla [4] has investigated the possibilities of **cultivating the lepra bacillus** for the purpose of **providing serum** for the treatment of the disease. He decided that the bacillus can be cultivated upon human blood-serum when the method of Herman is used, and that secondary cultures may be made upon the same media. Beef bouillon may be used also if it is covered with human blood-serum, and the culture may then be transferred to bouillon culture. In the latter cultures the bacillus is aerobic and motile. It is important that the temperature should be kept at about 37° C. and should never go above 45° C. The cultures should be neutral or slightly alkaline. He thinks that by using such cultures it may be possible to provide a serum for the treatment of the disease.

R. S. Woodson [5] reports a case of leprosy treated with **Calmette's antivenene.** The woman, who was 36 years old, was given 2 cc. daily at first, and this was gradually increased to 40 cc. During the first few weeks the case became acute, and had fever, eruption of tubercles, and general neuritis; but at the time reported, 3 months later, she had greatly improved, had no ulcerations or tubercles, the anesthesia was much diminished, as was the nasal secretion, and her general appearance was much better. She had, however, been getting large doses of hoang-nan at the same time, and this may have had some influence in the improvement.

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[1] Lancet, Sept. 16, 1899.

[2] Lancet, Mar. 3, 1900.

[3] N. Y. Med. Jour., Nov. 18, 1899.

[4] Wien. med. Woch., Mar. 31, 1900.

[5] Phila. Med. Jour., Oct. 28, 1899.

## GLANDERS.

Zaudy [1] describes a case of glanders which occurred in a man of 25 who was admitted in a typhoid condition. There was, however, a boggy swelling about the left temple and there were small nodules in the subcutaneous tissues; he afterward showed a swelling of the leg, the nodules increased in number and some of them suppurated. The diagnosis was made during life, and was confirmed postmortem by the presence of wide-spread glanders nodules in numerous tissues and by the discovery of the bacillus upon culture. The man had worked about horses, and careful examination of the horses showed that one had glanders. Particular attention is directed to the fact that this horse had not presented any special signs of illness, and the owner had not the least suspicion that the animal had any serious disease. There had been, however, some discharge from the nose.

## TRICHINOSIS.

G. Blumer and L. H. Neumann [2] describe a **family outbreak of trichinosis** which occurred in two Italian families in Albany, involving, in all, 9 persons. The source of infection was determined to have been sausages. The pork from which these sausages were made was cut into relatively large pieces, which were about 1 cm. in diameter, and the large sizes of the pieces of pork explains the fact that the trichinæ had lived through two cookings, the sausages having once been cooked in boiling lard and subsequently thoroughly fried. When examined microscopically, the pieces of pork showed in many instances numerous trichinæ. The cases did not show any preliminary gastro-intestinal disturbance and the onset was insidious. There was in most cases very little muscular tenderness, though there was some aching in the muscles. A prominent symptom in all the cases was the edema of the face. The general course of the cases strongly resembled typhoid fever, a rise in temperature being exhibited by all but the mildest cases. Complications of interest were endocarditis in 1 case, lung disease in 4 cases, the occurrence of spots simulating rose spots, and erythematous eruptions. A very unusual complication was a femoral thrombosis. In no case was tenderness of the heels noticed. Examination of the blood confirmed previous work on the question and demonstrated the existence of a **marked eosinophilia in every case.** The lowest percentage of eosinophiles in the beginning of an attack was 17; the highest was

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[1] Deut. med. Woch., May 24, 1900.

[2] Am. Jour. Med. Sci., Jan., 1900.

50.2. In several of the cases the number of the eosinophiles was greatly reduced when they passed from observation, about a month after they were first seen; in several, however, the eosinophilia was quite as high or higher at this time than when first seen. Leukocytosis was observed in each case, and the neutrophiles were relatively decreased, corresponding to the increase in the eosinophiles. In some cases there was a decrease in small mononuclear cells. The number of eosinophiles present seemed to bear no relation to the severity of the disease, but the degree of leukocytosis did bear a direct relation. From the examination of the blood itself the observers saw no reason to believe that neutrophiles were transformed into eosinophiles.

W. T. Howard, Jr.,[1] reports a fatal case of trichinosis **without eosinophilia,** but with large numbers of eosinophilic cells in the muscle lesions. The patient was a woman of 35 who had had fever, diarrhea, and vomiting, with pain in the abdomen and muscles, presenting the clinical picture of multiple acute myositis. Excision of a portion of the muscles was carried out, and the trichinæ were found. Blood examination showed slight leukocytosis, but no increase of eosinophiles. A differential count was not carried out, but the eosinophiles were not present in large numbers; a postmortem differential count of blood from the liver and spleen was carried out, however, and showed no increase in the eosinophiles. Bacteriologic examination showed no micro-organisms. Around the areas of infiltration there were large numbers of plasma cells and less numerous eosinophilic cells, and Howard states that the **development of the eosinophilic cells from the plasma cells** could be easily followed, the protoplasm of the plasma cells first becoming faintly and then strongly eosinophilic, and then showing fine granules, and later coarse granules, which were typically eosinophilic. The cells were at first mononuclear, some of them later becoming polymorphonuclear. He believes that most of the eosinophile cells were formed in the area of cellular infiltration about the trichinæ and in the muscles, and were not brought there from a distance. The cause was probably a chemotactic substance produced by the parasites or the degenerating muscle. There was certainly a marked chemotactic attraction for plasma cells. The eosinophilic cells were present in increased numbers in the gastric and intestinal mucosa, probably as a result of the recent presence of the trichinæ in the lumen and walls of the intestine. Bone-marrow was not obtained for study, but Howard states that he has recently observed the development of eosinophilic cells from plasma cells in two cases in the bone-marrow and in a number of instances in other organs. He criticizes Brown's statement that the eosinophilic cells in his cases probably arose from the neutrophiles. He believes that the only real argument in favor of this belief is that the neutrophiles were decreased, and thinks that a careful study of Brown's counts shows that the large mononuclear cells were present in decidedly great numbers, and it is from these cells that the plasma cells develop. He draws attention to the fact that eosinophilia is not a distinctive sign of trichinosis alone, as it has been reported in a number of instances of helminthiasis. [Howard's observations are interesting in connection with the origin of eosinophile cells, tending as they do to confirm the views of several investigators that these cells originate in other tissues than the hemopoietic.]

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[1] Phila. Med. Jour., Dec. 2, 1899.

## RABIES.

J. R. Bradford [1] gives a lecture on rabies based upon a study of the literature and of **between 300 and 400 cases.** He gives an interesting review on the susceptibility of various animals to the disease. All mammals are susceptible, as are birds, though the latter are not readily inoculated except experimentally. It is doubtful whether amphibia are susceptible. It is frequent in wild animals, and its occurrence in wolves and foxes constitutes a serious danger. It occurs in cattle, horses, and pigs; most commonly in cattle, very rarely in pigs. It is probably rare in pigs because they have so much subcutaneous fat. It is frequently found endemic in certain localities, and is hard to eradicate. It also occurs in epidemics. It is usually communicated by bites. The **cat is the most dangerous animal** because it flies at exposed parts and inoculates the victim directly, and, too, often bites the face, and inoculation in the face seems to be more dangerous than elsewhere, probably because the inoculation occurs so close to the central nervous system. From 10% to 20% of persons bitten by rabid animals acquire rabies. The variations in the occurrence of infection are largely due to the circumstances; as, for instance, if a man is bitten through his trousers, he is less likely to acquire the disease than when bitten directly on the hand. It has been acquired by postmortem inoculation and through the milk. The virus is found in the central nervous system and in the glands and their secretions, but not in the blood. It is destroyed by drying in about 2 weeks; by exposure to air and sunlight, in about 14 hours; in water, in from 20 to 40 days; and in carcasses, usually in from 3 to 6 months, though its presence in carcasses is often obscured by the presence of septic microorganisms which kill animals experimentally infected before the rabies virus has time to act. In the **diagnosis** it is extremely important to remember that the incubation period in human beings is almost always between 20 and 60 days, a longer incubation being extremely rare; one of a duration of 6 months or more is practically unknown. Bradford emphasizes the fact that it is unwise to kill an animal suspected of having rabies, for if observed for a few days the diagnosis may be readily established by the occurrence of death with the symptoms of rabies, while if the animal is killed and one trusts to experimental inoculation of other animals, no positive diagnostic results may be obtained, or, at best, the results are not positive until several weeks have passed because of the prolonged incubation period. In his second lecture he notes that at times it is impossible to make a diagnosis either by postmortem examination or by experimental inoculation. Clinically the disease in man shows a prodromal period, a second period of full establishment of the disease, and a third or paralytic stage. In the first period the characteristic symptoms are generally malaise, melancholy, sleeplessness, and a peculiar apprehensiveness, even if the patient does not know that he is in danger of rabies. In the second period there is severe and characteristic dyspnea, followed by convulsions, particularly of the muscles of deglutition; during the convulsions there is a peculiar gasping, which resembles, to the popular sense, the barking of a dog. The convulsions are peculiar in that they are excited by the slightest irritation, such as an attempt to drink, a draft, a sudden noise, and the like. There is fever, often glycosuria and albuminuria, and the apprehensiveness becomes intense. In the third stage there is paralysis, which begins in the limbs and involves the trunk. In some cases the disease is of purely paralytic type. In the dog the early symptoms are restlessness and the appearance of general melancholy, there is increasing excitement, and the animal finally becomes furious, bites everything near it, evidently has extreme hallucinations, and there is a peculiar long-drawn stridulous bark, dying away in a howl. The animal usually wanders away from home. In its excitement it swallows all sorts of foreign bodies. The disease in the dog lasts from 2 to 10 days, usually 4 or 5 days. The most characteristic features in the dog are the bark, the restlessness and excitement, and the difficulty in swallowing. The disease may be purely paralytic in the dog also. In the rabbit it usually is so, being characterized chiefly by somnolence, with weakness of the hind legs, which rapidly spreads to the other limbs and the trunk, and finally causes profound paralysis, while the appetite and the general health remain good. The postmortem changes are not characteristic, but in the dog consist in the discovery of large numbers of foreign bodies in the stomach with the absence of food and with scattered ecchymoses; these are somewhat characteristic. By experimental inoculation the diagnosis may be made in from 12 to 14 days, though it may take 40 days. The best method is subdural inoculation. As a rule, no general sepsis is caused. Bradford has examined 259 cases of suspected rabies in 4 years, and has found that the disease was present in 138, or 53%.

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[1] Lancet, Mar. 3 and 17, 1900.

Marks,[1] in a contribution concerning **hydrophobia immunity**, states that he discovered that certain animals that were injected with virus in the peritoneal cavity for other purposes were observed not to acquire hydrophobia. He therefore experimented with 41 rabbits, 4 guinea-pigs, 1 dog, and 1 goat, taking the cortex and basal ganglia of other animals who had had hydrophobia, emulsifying them in bouillon, filtering, and injecting each week 1 cc. of this emulsion. In no case did the virus, subsequently introduced beneath the dura, cause infection. The immunity was not due merely to brain-substance, because emulsion of normal brain did not produce immunity. Marks thinks it is possible, therefore, that a hydrophobia serum may be obtained in this way. Because of the wide-spread impression that animals that eat the livers of dogs that have died with hydrophobia do not acquire the disease,

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[1] Deut. med. Woch., Oct. 12, 1899.

Marks gave dogs extract of the livers of animals dead of hydrophobia, but found that this substance had no antitoxic effect.

## TUBERCULOSIS.

**Etiology.**—F. Hueppe[1] believes that too exclusive attention has been given to the **tubercle bacillus** in studying the etiology of tuberculosis. He insists that one must take into account the susceptibility of the subjects, that sanitary conditions must be improved, and that one should attempt to lessen the susceptibility of individuals by fostering proper physical development.

S. A. Knopf[2] directs attention to the **importance of tenement houses** in large cities **in the propagation of tuberculosis.** The bad air, poor drainage, and filth make the disease, when already present, practically incurable, and lead to further infection. In some instances as many as 20 consecutive cases of tuberculosis may be found in tenement houses within 4 years. Walking cases of consumption are most dangerous to the community, because they spread the disease in every direction by depositing their infectious sputum everywhere.

H. Walsham,[3] after a critical study of the literature, decides that **mitral stenosis and pulmonary tuberculosis** exist in the same individual so rarely that mitral stenosis can not be said to be conducive to the development of tuberculosis of the lung, though they are not antagonistic to each other.

W. Goldie,[4] in association with Sutherland and Young, has repeated the experiments of Flügge, and has again demonstrated that **colonies of tubercle bacilli** are **obtained upon plates** which are **held near the mouths** of patients with phthisis when they cough. The results of his experiments were as follows: 60% were positive in all cases in which the plates were used for 24 hours; 60% were positive when the plates were used for 24 hours by those whose cough was accompanied by expectoration; when the cough was not accompanied by expectoration and the plates were used for 24 hours, the results were positive in one-third of the cases; 28% of positive results were obtained when the plates were used but once during an attack of cough in the early morning; 14% were positive when the plates were used but once during an attack of cough in the evening. Goldie also believes that during coughing the bacilli are scattered about in the air and may remain suspended in the air for some time. The possibility of this he demonstrated by washing the mouth with an emulsion of Bacillus prodigiosus, and during the subsequent 5 minutes giving 12 coughs. The plates exposed during this time showed growths of the bacillus. Plates exposed in the air at intervals of 5, 10, and 15 minutes, and left exposed for 5 minutes, showed in all instances a varying number of colonies of the bacillus.

Volland,[5] in discussing the manner of infection in tuberculosis, insists that **scrofula,** in his belief, **is due to infection by ingestion** of bacilli, and it is likely to occur in children when they are crawling about on the floor, since they thus contaminate their hands with bacilli and then introduce them into their mouths. This method of infection should be carefully guarded against.

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[1] Berl. klin. Woch., Oct. 30, 1899.
[2] Jour. Am. Med. Assoc., May 12, 1900.
[3] Brit. Med. Jour., Oct. 28, 1899.
[4] Canad. Pract. and Rev., Aug., 1899.
[5] Berl. klin. Woch., Nov. 20, 1899.

Lemiére [1] considers that so-called conjugal contagion of tuberculosis deserves more properly the name of **marital contagion,** because in the great majority of cases it consists in transmission from the husband to the wife, while the opposite is exceptional. The reason for this is that the wife leads a sedentary life, is confined to the house, and is also exposed to the danger of pregnancy, labor, and lactation.

H. Roger and M. Garnier [2] described an important observation concerning the transmission of tuberculosis. It has been stated by a number of authors that the bacillus of tuberculosis is not found in human milk in cases of tuberculosis in nursing mothers, and it has come to be practically accepted that the bacillus does not appear in the human milk. They report, however, an instance in which the presence of the bacillus was demonstrated, animals injected with the milk dying of generalized tuberculosis. The mother had advanced pulmonary and pharyngeal tuberculosis at the time of the child's birth, and died 17 days afterward. The child had nursed at the mother's breast for 2 days. It died 6 weeks after birth, and showed tubercles in the liver, spleen, kidneys, and mesenteric glands. They consider that the tuberculosis arose chiefly, if not entirely, from **infection through the digestive tract by the mother's milk.**

J. G. Adami,[3] in discussing **bovine tuberculosis in Canada,** refers to the work of Rabinowitch and Kempner on the presence of tubercle bacilli in milk from animals whose udders are unaffected. Adami and Morton made postmortem examinations of 10 cows, and found no tuberculosis of the udder; and yet in 6 of these animals they had found bacilli in the milk which were considered undoubtedly tubercle bacilli, and which in one instance caused tuberculosis in a guinea-pig. Against the statement that these bacilli are not true tubercle bacilli Adami points out that our knowledge of the tubercle bacillus is not yet sufficiently definite to allow of a positive statement. In these cases the udders were carefully washed before milking, and if a bacillus resembling the tubercle bacillus was present, and not the tubercle bacillus itself, it must be a normal inhabitant of the larger milk-ducts. Also, there was a direct relationship between the number of the cells in the milk and the extent of the tuberculosis in the animal. Adami believes that he has shown that the cells of the liver and kidney can take up and discharge the colon bacilli, and that the bacilli in this passage tend to become attenuated; he believes that the same attenuation occurs in the tubercle bacillus. In these cases he does not believe that the milk containing such bacilli is of high infective power, and still considers that local tuberculosis of the udder is particularly to be guarded against. Concerning the prevalence of tuberculosis in Canadian cattle, he states that there is no large tract of country in the north temperate zone which is so free from bovine tuberculosis. In 1898 in suspected herds 10,000 head of cattle were inoculated with tuberculin and only 5% reacted, and in 90,000 head inspected in Montreal only 80 were rejected in all, and only 2 of these were considered to be suffering from tuberculosis. Also he has been unable to find an animal with disease of the udder in order to carry on some experiments which he has undertaken, hence such disease is rare. This rarity of the disease in Canada makes it seem to him possible to undertake experiments for its extermination. He does not believe that it is practicable to attempt its extermination at once throughout the whole Dominion, but suggests that the experiment should be begun by attempting to eradicate the disease in islands, as, for instance, in Prince Edward's Island.

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[1] V Congres de méd. interne, 1899.
[2] Compt. rend. de la Soc. de Biol., Feb. 24, 1900.
[3] Phila. Med. Jour., Dec. 30, 1899.

L. Rabinowitch,[1] in investigating the milk supplied by **8 dairies which furnished infants' milk** in Berlin, found that the milk from 3 dairies in which the tuberculin test was regularly carried out contained no tubercle bacilli, while 5 other samples from dairies where the test was only irregularly used repeatedly contained tubercle bacilli. She also found bacilli in 2 samples of sana, a preparation of cows' fat, and in several samples of kephyr made from milk which had not been pasteurized and that came from dairies where the tuberculin test was not used. She decides, therefore, that this furnishes sufficient evidence that it is **essential to use the tuberculin test** in dairies which supply milk.

H. E. Annett [2] reports his investigation of **28 samples of margarin,** 15 of which came from Berlin and 13 from Liverpool. In only one did he obtain tubercle bacilli. Margarin may be infected by the manipulations in its preparation, or from the butter and milk which are added to it, or from the cattle from which the fat is taken. That it may contain tubercle bacilli is shown by the fact that Morgenroth found these bacilli in 9 of 20 samples.

Weissenfeld,[3] in an examination of 32 specimens of **market butter** for tubercle bacilli, found that in 2 cases inoculated guinea-pigs showed genuine tuberculosis, and in 7 cases pseudotuberculosis developed. Artificial casein preparations were examined also, and all of them were found to contain greater or lesser numbers of bacilli.

J. A. Robinson,[4] after reviewing the literature upon the subject, reached the conclusion that there is no positive evidence of danger of transmission of tuberculosis from cattle to man by contagion or by eating tuberculous flesh, but that we must admit that there is **danger from the use of milk** from tuberculous cattle. He thinks that there should be greater tendency to teach the public to recognize the danger of acquiring tuberculosis from human beings rather than to emphasize too strongly the danger from cattle.

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[1] Deut. med. Woch., June 28, 1900.

[2] Lancet, Jan. 20, 1900.

[3] Berl. klin. Woch., Nov. 27, 1899.

[4] Jour. Am. Med. Assoc., Jan. 27, 1900.

E. Moore,[1] in considering the importance of **bovine tuberculosis** of the human species, belittles the influence of cattle in the transmission of the disease. He states that he has been unable to find any well-proved case of consumption that could be attributed to infection from tuberculous cattle, and several other statements are made in the same line, one of them being that tuberculosis is much more common in cities, while the tubercular products of affected animals are used fresh in the country. Letters are recorded coming from various sources which he uses in support of his views.

F. F. Friedmann [2] discusses his results in the investigation of post-mortem material and of tonsils obtained by operation in their relation to the question of **primary tonsillar tuberculosis.** Postmortem he observed 1 case in which there were tubercle bacilli and typical morphologic appearances of tuberculosis of the tonsils without any signs of disease elsewhere. There were also a number of other cases in which the tonsils were primarily affected. In a series of other cases typical morphologic appearances were found, but bacilli were absent; hence these cases could not be used in the discussion. In other cases in which he found tuberculosis elsewhere, but in which the tonsils were free from tuberculosis, smears from the surface of the tonsils showed the presence of tubercle bacilli. This is considered sufficient evidence that animal injections can not be used in demonstrating the presence of tonsillar tuberculosis. The material obtained by operation from living children showed nothing in 46 cases; in 6 cases there was swelling of the glands of the neck, but the tonsils showed no evidences of tuberculosis. In 1 case with enlargement of the glands of the neck there was possibly a tonsillar tuberculosis, but in only 1 case did he find actual evidence of tuberculosis of the tonsils, and this was probably secondary, because the child had signs of tuberculosis of the lung. Friedmann thinks that his cases show definitely that primary infection of the tonsils through the food may take place, and he believes that this is the most frequent mode of infection in primary tonsillar tuberculosis.

J. Wright,[3] in a discussion of the recent literature on tuberculosis of the faucial tonsils, insists that one is not justified in regarding the tonsils and adenoids as the only portal of entry for the tubercle bacillus in the pharynx. Concerning Lewin's results (5 % of latent tuberculosis of adenoid growths in 200 cases) he states that this is at most merely the percentage that is found in the locality from which the report comes. He thinks that Lewin's tables are also somewhat selective, and at best 5 % of tuberculosis of adenoids does not seem strikingly large considering the enormous frequency of tuberculosis in general. He also criticizes Lewin's results because he did not use animal inoculation, but depended merely upon microscopic examination. His own results from the examination of 54 faucial tonsils, 51 pharyngeal tonsils, and 16 lingual tonsils were in all cases negative microscopically. In 12 instances animal inoculation was negative in each case. He thinks that perhaps the difference between his results and Lewin's may be partly due to difference in locality, but believes that part of it is due to error. He considers that it is well established that these structures may be the seat of tuberculosis, but does not think that the frequency of such a condition has yet been satisfactorily shown.

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[1] N. Y. Med. Jour., Sept. 2 and 9, 1899.

[2] Deut. med. Woch., June 14, 1900.

[3] N. Y. Med. Jour., April 7, 1900.

B. Dembinski[1] discusses the **rôle of the leukocytes in experimental tuberculosis.** The inoculation of the healthy guinea-pig with tubercle bacilli results in a leukocytosis which is at first polynuclear, but after 3 days becomes chiefly mononuclear. Leukocytosis becomes marked after 24 hours. If the tuberculous guinea-pig is inoculated with cultures of tubercle bacilli, the leukocytosis shows a large number of mononuclear elements and is much more pronounced from the beginning than in previously healthy subjects; hence in tuberculous subjects inoculation of tubercle bacilli seemed to cause the cells to react much more rapidly than in healthy animals. When pigeons were inoculated with avian tuberculosis, leukocytosis was seen pronouncedly in the first few days; while if the human bacillus was inoculated, leukocytosis was not marked, but giant cells which inclosed large numbers of bacilli were seen after the second day. In the first case the leukocytosis was insufficient to arrest the spread of the disease, while in the second case the giant cells seemed to have much more phagocytic action.

**Symptomatology.**—Mircoli[2] directs attention to the **cases in which fever is absent.** Such cases are not very rare, and in experimental tuberculosis the rise in temperature does not occur if the dose injected is very small and the disease assumes a chronic course. However, in human subjects who have no fever, exertion, emotion, or any other form of disturbance readily causes a rise of temperature. The absence of fever in tuberculous infection Mircoli attributes to the fact that while the proteins of the tubercle bacilli raise the temperature, the toxins lower it, and in the apyretic cases the toxins have probably the more active effect.

Barbier,[3] in discussing the **temperature in pulmonary tuberculosis,** states that, as a rule, the periods of tuberculosis which are apyretic are in reality not free from fever; if the temperature is taken with proper frequency, it will be found that there are elevations of temperature. It is especially important that the temperature should be investigated at the times when rises are likely to occur. These periods are usually from 8 to 9 in the morning, and between 2 and 6 in the evening. The temperature sometimes reaches its greatest height about 10 in the evening or 4 in the morning. It is evidently very important to have the temperature taken frequently in cases of suspected early phthisis.

Carcassonne[4] discusses the **association of muscular atrophy with pulmonary phthisis.** The scapulothoracic muscles show the most marked atrophy, and there seems to be some direct relation between the evolution of the changes in the lungs and the atrophy of the muscles. He describes the condition as being most marked on the side affected if both lungs are involved, and as almost exclusively on the affected side if but one lung is diseased. He thinks that unilateral atrophy of the scapulothoracic muscles is an indication of some value of tuberculosis of the adjacent lung. He attributes the changes to alteration of the nerve-fibers of the affected pleura, and consequent degenerative changes in the cells of the cord, these changes involving the cells supplying the scapulothoracic muscles.

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[1] Gaz. hebdom. de méd. et de chir., Jan. 11, 1900.

[2] Gaz. degli Osped., Sept. 24, 1899.

[3] Gaz. des Hôp., Nov. 10, 1899.

[4] Arch. gén. de méd., Feb., 1900.

T. Campbell [1] reports another case of tuberculosis of the lungs in which there was a cavity in the left lung near the heart and the pulsation of the heart caused curious **crepitations and grating sounds synchronous with the heart-beat.** Subsequently there was a strange creaking sound which corresponded in time with the cardiac rhythm. The latter was often heard at some distance from the patient.

Crespin [2] reports a case of tuberculosis showing signs of cavity (cracked-pot sound, amphoric breathing, rales) in which typhoid fever occurred. Contrary to the customary experience under such circumstances, the **patient improved after the typhoid fever** had passed, the signs of cavity disappeared, and those of infiltration of the lung grew much less marked.

Rose [3] discusses **tuberculous pneumothorax,** particularly referring to the 19 cases which have been seen in the Bethany Hospital in 8 years. Within this time there were only 3 nontubercular cases of pneumothorax. Of the 19 cases reported, only 3 occurred in women. It was commonest in the third and fourth decads, and in this series was most frequent on the right side. Fifteen cases were fatal, while 3 apparently recovered, and 1 was discharged unimproved. Rose notes particularly that death did not occur on the average within the first month, as West states that it commonly does, only 7 of his cases dying within this time. Ten cases were purulent, 7 serous, and in 2 there was no exudate. As to the treatment of pyopneumothorax, Rose states that if there is absence of decided involvement of the lung and no marked complication, there is distinct prospect of cure; but he believes that operation should be carried out whenever practicable, whether cure is expected or not, as it often causes marked improvement, and may be done with only partial anesthesia and very little resulting shock. Of the 7 cases of hydropneumothorax, 2 recovered and 1 was discharged unimproved; of the 2 cases that recovered, 1 had decided involvement of the lung when discharged, and the other ultimately died of meningitis. The postmortem in the latter case showed that adhesions had formed over the lung surfaces and the hydropneumothorax was entirely cured. While discussing the cure of 1 case of serous pneumothorax in a patient with wide-spread phthisis, he notes the rarity of such a cure. He gives brief abstracts of the other cases of this kind already reported, which number but 10. In his cases of pneumothorax there were 2 without effusion; one of these occurred in a patient who had but slight disease of the lungs and a normal pleura, and this patient rapidly recovered. The other case which had no effusion was bilateral—an extreme rarity. The end came on with great rapidity, since both sides were affected within a short time of each other. Rose states that this is the fourteenth case thus far reported of a bilateral pneumothorax. He collects 12 cases of complete recovery from tubercular pneumothorax without effusion. In conclusion he states that the prognosis of pneumothorax varies greatly, and is dependent largely upon the nature of the effusion. It is likely to end in death in most cases, but the course may be protracted for months, or perhaps years, and death may finally be caused by the advance of the tuberculosis and not by a pneumothorax. Cures are exceptional, but do occasionally occur. The purulent cases are the most serious. The prognosis can not be satisfactorily settled early in the case.

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[1] Brit. Med. Jour., Nov. 18, 1899.

[2] V Congres français de méd. interne, 1899.

[3] Deut. med. Woch., Oct. 26 and Nov. 22, 1899.

Drasche,[1] in discussing **198 cases of tuberculous pneumothorax,** found that 158 occurred in men, 40 in women. It was right-sided in 55 %, bilateral in 3 cases. Of the latter 3 cases, one died instantaneously, the second after 2 hours, and the third after 12 hours.

Eisenbarth [2] describes a case of tuberculosis of the larynx in a man of 47 who had tuberculosis of the lungs and intestinal tract also. The laryngeal symptoms almost entirely disappeared, and at autopsy it was found that the ulcer of the **larynx was entirely healed.**

G. Mayer [3] describes an important case of **miliary tuberculosis** in a man of 21 which was apparently proved to be **secondary to tuberculosis of the skin.** The man had tuberculosis of the lungs also, but serial sections of the skin lesions showed that a rupture had occurred into a vein. Shortly before death a sudden decline of the temperature was observed and bacilli appeared in the sputum in the latter part of the attack.

Ménétrier [4] describes a case of pulmonary tuberculosis in which signs of **meningitis** appeared. The patient then developed a **phlegmasia** of the left leg, and at the same time the symptoms of meningitis disappeared rapidly, the thrombosis occurring as a critical phenomenon.

Rapia and Fortiveau [5] have carried out some investigations to determine whether **tuberculin is present in the urine** of the tuberculous. They injected guinea-pigs with tuberculin, and then in young guinea-pigs made intraperitoneal injections of the fresh urine and the urine after filtration. The results were very inconstant, but in some cases there was fever, and the animal died subsequently with the chronic symptoms produced by injections of tuberculin.

**Diagnosis.**—Daremberg and Chuquet [6] discuss the importance of the **inverse influence of fatigue and repose upon the temperature** of the tuberculous, both as a valuable element in early diagnosis and prognosis and as one that is of importance in treatment. They present a number of charts which show pronounced variations in temperature after walking or other slight effort in subjects who were tuberculous but showed few signs, and in whom the diagnosis was somewhat difficult. Two cases are contrasted, both being suspected of tuberculous infection, but neither showing signs in the chest sufficient for diagnosis. One showed always decided elevation of temperature after walking, while the other did not. The latter person recovered upon treatment with iron, while in the former almost entire recovery was obtained by rest, overfeeding, and counterirritation. They insist upon the necessity for observation of the temperature in women and in young girls at the time of menstruation if they are suspected of being tuberculous, and charts are presented to show the marked fever that is likely to occur at this time. They emphasize the fact that this fever shows that women with tuberculosis should usually be put to bed at the time of the menses.

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[1] Wien. klin. Woch., Dec. 21, 1899.
[2] Deut. Arch. f. klin. Med., Sept. 29, 1899.
[3] Münch. med. Woch., Jan. 23, 1900.
[4] Gaz. des Hôp., Jan. 19, 1900.
[5] Gaz. hebdom. de méd. et de chir., Oct. 25, 1899, p. 953.
[6] Rev. de méd., Sept. 10, 1899.

E. Fromm [1] has investigated the question as to whether the **ephemeral rises of temperature** often observed on the day patients are admitted to the hospital are related to any special form of disease. Of the 100 cases investigated in which this rise of temperature occurred, it was found that 49 were instances of disease of the respiratory apparatus, and almost all these were either phthisis, bronchitis, or pleurisy. The bronchitis and pleurisy might readily have been tuberculous. Of the other 51, about half were febrile gastro-intestinal disturbances or rheumatism. The rise of temperature was not frequently observed in other conditions; hence Fromm decides that while it by no means definitely indicates the presence of phthisis, it is a phenomenon which, when it occurs in otherwise afebrile cases, should arouse suspicion of the existence of phthisis, and is of some diagnostic importance in this connection.

P. D. Bourland [2] reports 4 cases of tuberculosis of the lungs to illustrate the variations in the early course of the disease and the possible difficulties in diagnosis. He especially insists that even in the absence of tubercle bacilli in the sputum slight local or general signs demand great care in the management of cases, and always should arouse suspicion of tuberculosis. He especially notes the frequency of gastric complaints in the early stages, and among local signs in the lungs insists that a **diminished respiratory murmur** and its significance are often overlooked or slighted. The diazo reaction he finds of importance in diagnosis, as it will often direct attention to the possibility of tuberculosis. Its intensity, also, is an index to the severity of the disease. He has noticed repeatedly that it intermits from time to time. The temperatures of the cases show an average range slightly higher than normal and a lack of definite rhythm.

S. G. Bonney,[3] in speaking of the early diagnosis of tuberculosis of the lungs, states that there is not sufficient appreciation of the almost pathognomonic importance of **circumscribed bronchiolitis,** even in the absence of other signs; and he also considers that examiners do not sufficiently often make use of cough followed by forced inspiration to elicit the presence of slight moisture in the finer tubes.

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[1] Centralbl. f. innere Med., June 30, 1900.

[2] Phila. Med. Jour., Dec. 23, 1899.

[3] Med. News, Sept. 30, 1899.

S. G. Bonney,[1] in discussing the early diagnosis of pulmonary tuberculosis, states his belief that there is too great a tendency to disregard the importance of various degrees of **hemoptysis** as an early sign. He thinks the x-rays of marked importance in diagnosis; and, among other signs, emphasizes the value of morning cough, slight daily fever, and marked constitutional disturbance. In studying 546 cases in his own practice he found that in 15% the disease seemed to have followed an attack of influenza, in 21% there was a bronchial onset, and in 20% he obtained a history of onset with hemoptysis.

H. Walsham[2] discusses the importance of **myoidema** in pulmonary tuberculosis. It is not common in the incipient stage, though it may be seen as fibrillary contractions. In more advanced stages it is very often observed. Usually the fibrillary variety is present, but frequently the nodular form may be seen. In the advanced stage of phthisis the nodular form may practically always be elicited, unless there is very advanced wasting of the pectoral muscles. It is practically only an indication of the condition of the muscular system of the subject. It occurs more frequently in phthisis than in any other disease, but it is by no means diagnostic of this condition.

T. F. Harrington[3] considers decided **dilation of the pupils** an important suggestive sign of tuberculosis. He does not think that it by any means indicates positively that tuberculosis is present or will develop, but he has very frequently observed it in persons apparently normal, and in others with typhoid fever, pneumonia, and other conditions, who afterward developed tuberculosis. He has come to look upon it as strongly suggesting the possibility of the future development of tuberculosis.

Moncorv[4] claims that a **diagnosis** may be made **between malarial fever and tuberculosis** by **applying guaiacol externally.** In tuberculosis a fall in temperature occurs, but this is not seen in malaria. The two diseases are frequently associated in the tropics, and both are common. The diagnosis is often difficult.

M. Henkel[5] describes satisfactory results in the **diagnosis of tuberculosis by aspiration of the lung** with a hypodermic syringe and the examination of the fluid obtained for tubercle bacilli. The method is stated to be useful in cases in which there is no sputum, and Henkel believes that it is justifiable in cases in which there are strong suspicions of tuberculosis. The puncture should be made at the point where the physical signs are most marked.

A. Girard[6] describes the following **method** as a satisfactory one **for demonstrating the presence of tubercle bacilli** in the sputum and for studying the histologic elements of the sputum. He places a portion of the sputum in about 10 times its volume of a solution of hypochlorite of soda of about $\frac{1}{3}$ of 1 % strength, which is colored by the yellow chromate of potash. It is allowed to settle in this solution for 24 hours, or is centrifugated after about 15 minutes. A few drops of normal potassium or sodium solution are then added to transform the hypochlorite into the chlorid ; distilled water is added to the mixture, it is centrifugated, and the water is poured off. The deposit is then easily examined and bacilli are readily demonstrated.

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[1] Med. News, Sept. 30, 1899.

[2] Lancet, Jan. 27, 1900.

[3] Phila. Med. Jour., May 28, 1900.

[4] Bull. Acad. de méd., Sept. 19, 1899.

[5] Münch. med. Woch., Mar. 27, 1900.

[6] Jour. de méd. de Par., May 13, 1900.

Strasburger [1] recommends the addition of 2 parts of 96 % alcohol to 1 part of the fluid examined in looking for tubercle bacilli in urine or other fluids or in feces. After mixing with alcohol he centrifugates or allows the fluid to deposit its sediment. The value of the method, he states, consists in the fact that the bacteria are of decidedly greater specific gravity than the alcohol mixture, and hence they deposit readily. He considers this method particularly valuable in examining the feces.

L. Rabinowitch [2] reports the discovery in the sputum of a case of gangrene of the lungs of a bacillus which had the morphologic and staining characteristics of the tubercle bacillus, but differed from it in its cultural characteristics and in the fact that it did not cause tuberculosis when injected into guinea-pigs or mice. This seems to be the first instance of the discovery of a bacillus with the general microscopic characteristics of the **tubercle bacillus but without** its **pathogenic properties** in the sputum. It is possible, however, that other instances will be discovered if looked for, though such cases must be very rare. In general clinical work there is little danger of confusion in staining for tubercle bacilli because of the rarity of such bacilli.

E. Levy and H. Bruns [3] discuss the early diagnosis of tuberculosis of the lungs and the **importance of animal experiments.** They do not believe that it is possible always to make the diagnosis by any one method, but find intraperitoneal injections of the suspected sputum, after carefully washing it in sterile salt solution, one of the most satisfactory methods. Of course, unless there is ulceration or softening there will be no results from the sputum injections ; they will, however, often establish the diagnosis. Levy and Bruns have demonstrated that extremely minute quantities of bacilli will produce tuberculosis in guinea-pigs. They diluted the washed sputum with bouillon to such a point that the injections contained as little as $\frac{1}{400000}$ cc. of sputum. Nevertheless generalized tuberculosis resulted in guinea-pigs injected with this amount. In order to avoid the occurrence of purulent peritonitis in the cases injected they have sometimes heated the sputum to 60° C. for 10 minutes and injected animals with this. It is often valuable, however, to determine the existence and character of a mixed infection.

Brieger and F. Neufeld [4] report a series of cases in which tuberculosis was shown to be present by positive reaction to tuberculin, and yet repeated examination of the sputum showed **absence of tubercle bacilli.** Some of them were cases of mixed infection and some were not. The authors insist that it is of great importance to recognize such cases and to have them properly isolated from other bronchial infections not tuberculous, as there is, of course, great danger that the tuberculosis would be communicated to the others if tuberculous cases were placed in the same ward. They insist that the mere absence of tubercle bacilli by no means constitutes final evidence that the case is not tuberculous, even when the appearance of the case is such that one would expect that tubercle bacilli would be easily found if it were tuberculosis.

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[1] Münch. med. Woch., April 17, 1900.

[2] Deut. med. Woch., April 19, 1900.

[3] Deut. med. Woch., Mar. 1, 1900.

[4] Deut. med. Woch., Feb. 8, 1900.

Brieger, [1] in discussing the treatment of tuberculosis with tuberculin and similar substances, begins his article with the statement that **tuberculin** is extremely important **in the diagnosis of tuberculosis** in man and animals. The importance of the tuberculin reaction is demonstrated by the fact that it is positive in cases that show no fever and have the slightest kinds of lesions. It allows one to diagnose between tuberculosis and diseases closely resembling it, particularly those chronic conditions caused by the influenza bacillus. That it is sometimes not positive in advanced tuberculosis is not surprising, since the organism is often so saturated with tuberculin that the slight addition made by the injection causes no further specific reaction. He refers to the important results which have been obtained in tuberculin injections in animals. In his extensive experience he has never seen any unfavorable results, and this is entirely in accord with the general experience of recent years, the suppositious bad results not being proved. The care which must be taken in judging of such supposed bad results is demonstrated by a case which he reports: A man, 30 years old, was sent to the institute with the desire to have a tuberculin test made. The temperature was not normal, and tuberculin reaction was not undertaken. On the third day after the patient entered the institution the temperature went to a high point, and within 2 days the man died of a miliary tuberculosis. If the rule had been overstepped and the man injected before he had been carefully watched for a time, there would have been every reason to have thought that the tuberculin had caused the final trouble; and it is quite probable that in some cases in which bad results have been reported the effects have been due to the disease itself and not to the tuberculin.

E. O. Otis [2] states that his observations of the use of the **tuberculin test in 111 cases** have convinced him that the test is not harmful. He finds that it is a perfectly feasible plan of diagnosis in out-patient clinics. He recommends that the injection be made deeply into the muscles. He considers that one should always use the same preparation of tuberculin, and one of standard strength. In early cases one should depend upon the local reaction, while in late cases, if the general reaction fails, the local reaction should be looked for. [Our own method has been to give small doses gradually increased; and in no case has there been an untoward result.]

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[1] Bericht über den Kongress zur Bekampfung der Tuberculose als Volkskrankheit, Berlin, 1899.

[2] Jour. Am. Med. Assoc., Oct. 28, 1899.

Grasset and Vedel [1] discuss the **results secured** in the **diagnosis** of tuberculosis by injections of tuberculin. Fourteen cases which they report were negative; in 6 of these it was possible to determine the nature of the disease, and it was found that all were instances of diseases other than tuberculosis; 2 were syphilis, 3 of the negative cases were advanced phthisis. In 14 cases positive results were obtained, and these cases, so far as is known, were all tuberculosis.

B. Fraenkel [2] gives a general review of the value of tuberculin in the diagnosis of tuberculosis. He states that in but 1 case has he met with an apparent example of reaction when the patient did not have tuberculosis. This was in a case of nasal ulceration, in which a diagnosis between lupus and syphilis was in doubt. A general reaction was obtained with 1 mg. of tuberculin, but no local reaction was seen. Antisyphilitic treatment produced a cure of the ulceration, but the patient still reacted to 1 mg. of tuberculin, and it was evidently a **combination of tuberculosis and syphilis.** He thinks that tuberculin injections should be made in all cases in which ordinary methods are insufficient for a diagnosis, and in all those diseases in which there is doubt as to the existence of tuberculosis. In the latter class he particularly mentions cases of chlorosis or those which resemble chlorosis, in which there is indistinct indication of lung trouble; doubtful cases of scrofulosis in children; and surgical tuberculosis.

E. L. Trudeau,[3] in a general discussion of vexed questions relating to tuberculosis, insists upon the value of the tuberculin test in diagnosis; he does not consider that it is dangerous or that it has been proved that harm has resulted from its use. Small, graded doses should be used at intervals of two or three days. He has also found the x-rays of value in the diagnosis of incipient disease. The attempts to provide a serum for tuberculosis have been practically failures. In Trudeau's laboratory it has been discovered that attenuated living cultures of avian bacilli inoculated into rabbits increase their resistance to virulent mammalian cultures, and cultures of bacilli attenuated by years of growth increase the resistance of animals also, but the immunity produced is only relative. Trudeau found that tuberculin made from the fluid of a culture attenuated by long growth is as useful as tuberculin from virulent cultures. He believes that adenoid growths are frequent portals of infection for tuberculosis. He makes a number of suggestions for research concerning tuberculosis.

O. Brieger,[4] in discussing the importance of hyperplasia of the tonsils in the development of tuberculosis, states that there are serious objections to the use of **tuberculin injections** to determine the presence of **latent tuberculosis.** He considers that Trautmann's hypothesis concerning the relation between hyperplasia of the tonsils and tuberculosis is unsatisfactory because based entirely upon the results of tuberculin tests. Brieger considers that tuberculin is usually of value for the demonstration of the nature of latent tuberculosis of the mucous membranes; but certainly the test with tuberculin for latent tuberculosis of the tonsils gives much higher positive figures than microscopic investigations of incised tonsils would indicate to be correct. It is quite true that children with hyperplasia of the tonsils give a local and general reaction to tuberculin very frequently. When one examines these tonsils microscopically, however, usually, and in Brieger's experience in every case, one finds one's self unable to discover any evidence of tuberculosis. This is partly attributable to the fact that latent tuberculosis in children is an extremely common thing, and that the general reaction is due to disease situated elsewhere; the local reaction always has to be considered carefully, because hyperemia and swelling of the tonsils is so common in children that it is not at all surprising that it is frequently observed at the time of the tuberculin injection. The use of tuberculin for diagnosing latent tuberculosis of this form must be undertaken with foresight, and the results should be considered carefully before conclusions are drawn.

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[1] V Congres français de méd. interne, 1899.

[2] Berl. klin. Woch., Mar. 19, 1900.

[3] Johns Hopkins Hosp. Bull., July, 1899.

[4] Bericht über den Kongress zur Bekampfung der Tuberkulose als Volkshrankheit, Berlin, 1899.

E. Fuerst[1] reports an interesting and important case of **hysteria** in which tuberculosis of the lungs had been suspected because of fever, pain in the chest, and cough, with bloody expectoration. An **injection of tuberculin caused a decided rise in temperature,** but there were curious symptoms associated with it. Hence, suspecting hysteria, an injection of distilled water was given, and the same febrile reaction was obtained. This was also true later after the mere introduction of the hypodermic needle. The importance of this result is evident, and leads Fuerst to recommend that one should always try control injections of distilled water before coming to a positive decision in doubtful cases, particularly if there is any suspicion of hysteria.

Leithwood[2] carried out a series of investigations concerning the value of **tuberculin** as a test for **tuberculosis in cattle.** Two herds of cattle were injected, one consisting of 52 cows and 2 short-horned bulls; the other of 16 cows and 1 short-horned bull. Fifty animals were found free from tuberculosis, 17 were declared infected, and 4 were doubtful. Examination of the udders of the animals that reacted showed only one instance of induration. Tubercle bacilli were found in the milk but once; 2 of the 3 bulls were found tuberculous; and after 10 of the animals were killed, lesions verifying the results were found in every case. One important point of the investigations was that it was shown again, as has been noted before, that repeated injections of tuberculin will cause the reaction to disappear in tuberculous animals; it is insisted that tuberculin may be used by owners of cattle in this way for the purpose of concealing tuberculosis, and that it should be sold only to duly accredited persons.

E. Bendix[3] has carried out the **serum reaction** in tuberculosis in 40 persons. Three normal persons gave no reaction, 2 of them with undiluted serum, and the third, while reacting with undiluted serum, gave no reaction in a dilution of 1 : 3. A number of diseased persons who were not tuberculous gave no reaction with undiluted serum. Thirty-six cases of tuberculosis gave positive reaction except in 2 instances. Both these were rapidly progressing phthisis, and Bendix states that he noticed regularly that the intensity of the reaction was in inverse relation to the rapidity of the progress of the disease,—in other words, to the severity of the disease,—mild cases often reacting in a dilution of 1 : 50, while in severe cases the reaction was often seen only in a dilution of 1 : 5. In 1 case, when running a mild course, the reaction was seen in 1 : 30; as the course became much more rapid, it sank to 1 : 15; hence in this case a form of anti-agglutin seemed to have been produced. He considers the reaction of extreme importance in the early diagnosis of phthisis. Maragliano's serum had a markedly agglutinative action, while the old tuberculin had no such effect.

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[1] Deut. med. Woch., April 5, 1900.

[2] Lancet, Feb. 10, 1900 (Editorial).

[3] Deut. med. Woch., April 5, 1900.

M. Beck and L. Rabinowitch[1] have investigated the serum reaction for tuberculosis, following the directions given by Arloing and Courmont. In all, 73 persons were tested. Of 17 persons with beginning tuberculosis of the lungs, only 6 gave the reaction; of 16 with advanced tuberculosis, but 4 gave a reaction; 5 suspicious cases that reacted to tuberculin showed serum reaction in only 1 instance. A case of tubercular meningitis was negative; 1 of 2 cases of lupus was negative, while a case of healed tuberculosis accidentally discovered postmortem had given the reaction during life. On the other hand, 2 of 3 cases of croupous pneumonia, 2 of 4 cases of exudative pleurisy, 2 of 3 cases of bronchitis, 1 of 3 cases of rheumatism, 1 case of liver cirrhosis, and 1 healthy person gave the reaction. They also found the reaction very variable in animals infected with tuberculosis, and found that serum infected with other organisms and then centrifugated sometimes reacted. They decide that the reaction is not a specific reaction of tuberculosis, that it frequently does not occur in tuberculosis, and that hence it can not be used in the diagnosis of tuberculosis. [The description given of the cultures shows distinct variations from the descriptions given by Arloing and Courmont; it is therefore possible that the difference in results may be caused by the difference in the cultures used. At any rate, there is a curiously wide discrepancy between these results and those obtained by Arloing and Courmont and by other observers. The reaction is certainly of questionable value, but the results published by Beck and Rabinowitch do not by themselves demonstrate that the reaction is valueless.]

Combemale and Mouton[2] recommend the use of **artificial serum in the diagnosis** of tuberculosis. The serum is composed of sodium chlorid 7 parts, sodium phosphate 1 part, distilled water 1000 parts, 20 cc. of this being injected in the flank, and the temperature being observed every hour afterward. A rise will occur if the patient is tuberculous. They consider that the reaction is strong evidence of the existence of tuberculosis. [This method of diagnosis has been used by several others before, and the conclusion reached was that while tuberculous subjects do usually react, reaction frequently occurs in other conditions, and hence the method is of little value.]

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[1] Deut. med. Woch., June 21, 1900.

[2] Gaz. hebdom. de méd. et de chir., Jan. 25, 1900.

**Prognosis.**—M. Michaelis [1] adds some further observations concerning the **prognostic value of the diazo reaction** in tuberculosis. Besides his previous observations, he and Flamand have investigated 61 further cases, making 725 separate examinations. Twenty-five cases were negative; and of these, 3 were cured, 18 improved, 3 unimproved, and 1 died. Thirty-six gave a positive reaction; of these, none were cured, only 7 improved, 1 disappeared, 6 were unimproved, while 32 died. Of the total of 167 cases which he has so far reported, there were 56 with a negative reaction; and of these, 5 were cured, 44 improved, 5 unimproved, and 3 died; while of the 111 cases with positive reaction, none were cured, 15 improved, 3 disappeared, 13 were unimproved, and 80 died. The diazo reaction appears, therefore, to be a strikingly good index to the prognosis. Michaelis gives an unfavorable prognosis if the diazo reaction is positive.

W. Naegelsbach and Schroder,[2] in discussing the diazo reaction in cases of phthisis, report positive results in 12 of 110 cases. It is an **unfavorable prognostic sign, but is not always present** in cases which run a bad course. It therefore can not be positively depended upon in prognosis, though it does indicate an unfavorable prognosis.

L. Brieger,[3] in discussing the diagnostic and therapeutic value of the presence of tubercle bacilli and other bacteria in the sputum, speaks particularly of the **importance of a mixed infection.** Mixed infection with the influenza bacillus makes the prognosis much worse; pus cocci also increase the gravity of the prognosis; Bacillus pyocyaneus has a destructive action similar to that of pus cocci; the tetracoccus makes the prognosis very unfavorable. He insists upon the necessity for proper antiseptic care of the mouth in preventing mixed infection. In treatment of the cases he has found aromatic oils, particularly oil of peppermint, if used with care as to the secretion of the kidneys, of value in many forms of mixed infection. It must always be remembered that tuberculosis of the lungs may at any time be transformed into "pulmonary phthisis," and that this is the result of the mixed infection.

H. Walsham [4] describes a case of tuberculosis of the lungs in which the first evidences of consolidation of the lungs were provided by **skiagrams**; the same method of examination was also used in studying the progress of the disease, and Walsham believes that the x-rays provide a useful method of establishing the prognosis and determining the rapidity of involvement in these cases.

**Treatment.**—J. F. Russell [5] gives an interesting report concerning the **results of dispensary treatment** of walking cases of phthisis in the New York Post-Graduate Hospital. Between March, 1898, and July, 1899, there were at least 10 cases in which apparent cure was secured by the treatment instituted. It did not interfere with the patients' work, hence they remained self-supporting and did not become State charges. It consisted in having the patients report twice daily at the hospital, including Sundays and without regard to the weather, in order that they might receive a preparation of predigested fats to improve their nutrition, and, more important, that they might be kept well under control and be well instructed in details of hygiene, etc. They were carefully watched and examined, and kept interested in continuing the method of treatment. The other chief details were careful regulations as to clothing, moderate exercise in the open air, avoidance of late hours, sleeping with open windows in all weather, and instruction in minute details of hygiene. Russell believes that the results have been decidedly encouraging, and that there should be some general alterations of the methods of treating dispensary cases of phthisis, since they ordinarily pursue an unfavorable course.

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[1] Berl. klin. Woch., Mar. 26, 1900.

[2] Münch. med. Woch., Oct. 10 and 17, 1899.

[3] Berl. klin. Woch., Mar. 26, 1900.

[4] Lancet, July 15, 1899.

[5] N. Y. Med. Jour., May 5, 1900.

Richet [1] describes results which he has obtained in conjunction with Héricourt in investigating the effect of **exclusive meat diet** upon experimental tuberculosis. He found that dogs injected with cultures of tubercle bacilli died of tuberculosis ordinarily within 30 to 50 days; but if they were given exclusive meat diet, they often survived for over a year, and in some cases recovered entirely.

Héricourt and Richet [2] refer to their previous experiments, and now report that their investigations have led them to decide that the **muscle plasma**, and not the pulp of the beef, is the part which has the favorable influence. They prepare muscle plasma by mincing the beef, adding water, and expressing. This can readily be given to phthisical patients even when the stomach is unretentive, and they consider it valuable in the treatment of phthisis.

J. Cobb [3] discusses the **arid region of the United States** and its relation to the treatment of phthisis. He does not think that there is much difference in the climates of Arizona, Colorado, New Mexico, and western Texas, so long as the patient seeks an elevation above 2000 feet. If there is tendency to hemorrhage or if the disease is advanced, and usually if the patient is a woman, a medium latitude should be advised. Cobb advises that the patient should carry with him many comforts, as proper facilities for making people comfortable are absent in many regions, especially in the small towns. He gives a directory of the hotels in the more important towns.

C. F. Gardiner [4] recommends the **Colorado climate** in the treatment of consumption, and particularly insists that the patients should be exposed to the air for practically the whole 24 hours each day. This he accomplishes by the use of a special tent which has a hole at the top and sides raised somewhat from the floor, so that the air circulates from top to bottom.

A. Hillier [5] gives a discussion on the open-air treatment of phthisis, in the course of which he states that he has found **ocean voyages** of very **doubtful value.** While at times they do good, they also at times do decided harm, and are by no means to be recommended lightly.

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[1] Jour. des Praticiens, Dec., 1899.

[2] Rev. Scient., Mar. 10, 1900.

[3] Med. News, Oct. 21, 1899.

[4] Med. News, July 22, 1899.

[5] Practitioner, Aug., 1899.

V. Y. Bowditch,[1] in discussing the results obtained at the Massachusetts State Hospital for Consumptives in the year 1899, records that 214 cases were treated, of which 114 remained long enough to give opportunity for judgment as to the results. In 35 the disease was thought to have been arrested. In these cases the average residence in the hospital was 4½ months, and the patients averaged 15⅗ pounds gain in weight. The percentages are about the same as those obtained elsewhere, and the methods in general are similar to those usually described.

W. Calwell,[2] in considering the use of **drugs in phthisis,** reports that comparative use of simple tonic treatment for 1 year, followed by creasote treatment the next year, later by petroleum emulsion, and later by guaiacol for several months, in the same institution, showed no superiority of one treatment over another; but in the last year the open-air treatment was used, and he was so strikingly impressed with its effects as to lead him to compare it with the results obtained by using mercury and potassium iodid in syphilis.

W. Porter,[3] in discussing the treatment of the tuberculous, recommends the frequent use of **cardiac tonics** and keeping the bowel well emptied. He believes that this prevents absorption of poisonous matters from the intestines, and thinks that the useful results from creasote and its derivatives are probably largely secured in this way.

Schaper,[4] in discussing the **results** of the treatment of tuberculosis **in the Charité Hospital** in the last 10 years, shows that the mortality has fallen from about 54% to a little above 34%. These results he attributes to the fact that patients come to the hospital much earlier, and the disease is recognized in earlier stages, and to the improvement in the hospital facilities for treating the disease. Contrary to these results, the records of the Institution for Infectious Diseases in Berlin show a constant slight increase in the mortality. This is probably the result ot the character of the cases treated in this institution.

G. Champion,[5] in discussing the treatment of pulmonary tuberculosis by **immobilization of the thorax,** states that this treatment should be used in conjunction with a hygienic and dietetic treatment, and that when so used it is extremely valuable. It relieves many of the symptoms, particularly the cough and the intercostal pains, and causes the temperature to fall so that medicinal treatment does not cause any disturbance of digestion. It is not to be recommended in the emphysemas nor in tuberculosis when the disease is advanced; in other words, it may be harmful in cases in which the respiratory capacity is poor.

E. de Renzi and G. Boeri [6] report good results from **thiocol-Roche,** the potassium salt of sulphoguaiacolic acid, which contains about 52%

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[1] Boston M. and S. Jour., Feb. 8, 1900.

[2] Practitioner, July, 1899.

[3] Jour. Am. Med. Assoc., Mar. 3, 1900.

[4] Berl. klin. Woch., Mar. 19, 1900

[5] Thèse de Paris, 1899.

[6] Deut. med. Woch., Aug. 3, 1899.

of guaiacol; it is a white powder, not unpleasant to the taste, and is easily soluble in water. It may be readily taken in orange syrup. The best dose is from 15 to 30 grains daily. The cases reported showed very decided improvement in the general condition and in most symptoms, and the bacilli decreased largely in number. Metabolism experiments showed some increase of the uric acid while taking the drug, and there was some increase also of the acid sulphates and the neutral sulphur in the urine. The authors think that the increase of the uric acid indicated an extension in the oxidative processes of the body. [There was apparently no control of the amount and character of food taken, and the uric acid did not increase, on the average, more than 100 mg. a day. This can not be taken to be indicative of anything definite. The statement is made that the patients' appetites improved notably with the use of the drug, and the additional amount of food might be entirely sufficient to explain the increase in uric acid.] Frieser[1] reports good results from the use of thiocol in 19 cases of phthisis. He also observed marked improvement of the digestive symptoms.

A. Macgregor[2] reports that he has used **chenosol** in over 100 cases of pulmonary tuberculosis in doses of from 3 to 5 gm. 3 times a day. He believes that his results from this drug were better than those from the use of either guaiacol or creasote.

Guerder[3] has used injections of the **glycerin extract of fresh cod-liver,** completely neutralized and sterilized, in the treatment of tuberculosis. He found that it caused general improvement, with relief of the sweats and of the feeling of oppression, and that in guinea-pigs experimentally rendered tuberculous it to a considerable extent protected against the advance of the disease.

Aucean[4] reports a series of observations on the use of **vanadium** in the treatment of tuberculosis. He believes that the drug is a valuable tonic and stimulant of the appetite and digestion, and is of very great value in early stages of tuberculosis. The best manner of giving the drug is to administer it for from 3 to 5 days, and then to allow an interval of about the same time to pass before resuming it. The best form to use is the vanadiate of soda, given in doses of about $\frac{1}{30}$ of a grain.

Fraenkel[5] has gone over the literature of the treatment of tuberculosis by **cinnamic acid,** and records a large number of cases in which this has been used. In spite of the enthusiastic recommendation of Landerer, Fraenkel can not see that the records show any better results by this method of treatment than by the older methods. In 150 cases of tuberculosis of the lungs and in 5 of lupus in which Fraenkel has used this treatment, he has found no improvement which could be directly attributed to the drug, and in the pulmonary cases there seemed to be some increased tendency to hemorrhage. He also made experimental studies of the effects of the drug by injecting bacilli into the anterior chamber of rabbits' eyes and subsequently injecting cinnamic acid. No effect could be seen.

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[1] Merck's Arch., Sept., 1899.

[2] Lancet, July 8, 1899.

[3] Jour. de méd. de Par., Mar. 18, 1900.

[4] Thèse de Paris, 1899.

[5] Deut. Arch. f. klin. Med., Feb. 6, 1900.

N. Azmanova[1] discusses the treatment of phthisis by the use of **cinnamate of soda.** The results which she describes are not favorable. If the injections cause a rapid reduction of temperature, this may be considered a good prognostic sign. The use of cinnamate of soda is contraindicated in rapid forms of tuberculosis. In the period of infiltration and of softening the treatment often causes improvement, but in the acute and subacute forms it is likely to be harmful.

B. Alexander[2] gives a further report on his method of treating tuberculosis of the lungs with subcutaneous injections of **camphorated oil.** He has reduced the dose to 0.01 gram of camphor per day. Used after this manner, he has had excellent results, and is convinced of its value. Criegern[3] has investigated the value of Alexander's camphorated oil treatment of phthisis. He concludes that it is contraindicated in nephritis and when there is a tendency to hemorrhage. He had no satisfactory results, and does not think it is better than other balsamic preparations.

Flick[4] has had useful results from inunction of **europhen** in tuberculosis.

T. J. Mays,[5] in a further report upon the use of **silver nitrate injections** in the treatment of tuberculosis, states that while previously he has recommended that the injections should be given on the side of the neck corresponding to the infected lung, he now feels that it will oftentimes give better results to inject on both sides, or on the opposite side. He makes the statement, which seems somewhat fantastic, that there was a definite relation in some cases between the gain in weight and the use of injections on one side or the other.

M. Einhorn[6] has used **heroin** in tuberculosis, with relief of the cough; in asthma, with good effect upon the dyspnea and cough; and in chronic bronchitis and cardiac disease, with similar effects. He also found it useful in relieving the pains of cancer of the stomach, as well as in hyperesthesia of the stomach and gastralgia. J. R. L. Daly[7] finds heroin very valuable in the treatment of the **night-sweats** of phthisis. It does not seem to have any influence upon the temperature. In some cases it causes constipation, and Daly has known it to produce nausea. [We have found that it caused nausea, loss of appetite, and constipation in a number of cases.] W. R. Thomson[8] describes 2 cases in which heroin excited continued vomiting. In one case the heroin was thought to be the indirect cause of death, because of the exhaustion produced by the vomiting.

Bariè[9] has used **sodium tellurate in the night-sweats** of phthisis for 5 years without seeing any bad effects except a little colic or diarrhea. He gives it in a dose of 4 or 5 grains a day, and has had useful results.

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[1] Thèse de Nancy, 1899.

[2] Münch. med. Woch., No. 9, 1900.

[3] Berl. klin. Woch., Oct 23, 1899.

[4] Phila. Med. Jour., Oct., 1899.

[5] Phila. Med. Jour., Dec. 30, 1899.

[6] Phila. Med. Jour., Oct. 28, 1899.

[7] Boston M. and S. Jour., Feb. 22, 1900.

[8] N. Y. Med. Jour., Feb. 3, 1900.

[9] Jour. des Praticiens, Feb. 17, 1900.

Chalmonski [1] describes a case of **hemoptysis** in tuberculosis of the lungs in which he used **potassium iodid** with successful results. He believes that this drug might well be used for this purpose more frequently in cases with increased blood pressure.

Shepp [2] reports good results in the treatment of tuberculosis with **fluoroform.** The dose he recommends is from 4 to 5 drams a day, though more may be given safely. Fluoroform is a gas, soluble in water, and practically tasteless and odorless.

J. L. Green [3] recommends the **inhalation of formalin** as a preliminary to the open-air treatment of tuberculosis, believing that cases do much better when they have been started on this treatment. The preparation of formalin he uses contains 1 dram of this substance, with 4½ drams of glycerin, in 5 ounces of water, used in an inhaler. If the mucous membranes are sensitive, he adds 10 minims of aromatic spirits of ammonia to the mixture.

H. M. Thomas,[4] in discussing the treatment of pulmonary tuberculosis, states that he has seen proof microscopically that **vaporized drugs** may penetrate the pulmonary alveoli. In carrying out the treatment with inhalations he recommends an abundance of air compressed to 36 pounds to the square inch, which should be passed through a series of chambers to purify it and should be warmed in cold weather. If cough occurs, the treatment should be stopped. The most satisfactory face mask is funnel-shaped and composed of pure rubber, and fits the face thoroughly. He describes 50 patients in whose treatment various drugs were used in this way with satisfactory results. G. A. Evans [5] has had good results from the treatment of phthisis with inhalations of compressed air, and has found that a spray of crystallized carbolic acid in solution in glycerin-water is a valuable antiseptic.

Desprez [6] reports that he has used with success **inhalations of chloroform** in the treatment of tuberculosis, believing that the chloroform penetrates to all parts of the lungs in amounts sufficient to influence unfavorably the growth of the bacilli, and perhaps to kill them. He states that in a future publication he will give a more extended communication concerning the method of using chloroform. [In the consideration of the various forms of inhalation treatment that have been recommended it is well to remember that the lesions of pulmonary tuberculosis are but slightly exposed upon the mucosa of the bronchi, and, further, that their vascular character prevents the volatile antiseptics from penetrating the diseased areas.]

Pinquet [7] considers that the removal of the exudate in serous pleurisy often seems to hasten or cause the development of tuberculosis. This apparent tendency has been explained by considering that the exudate on the surface of the pleura contains many tubercle bacilli, and when the two surfaces are brought together, the friction is likely to cause invasion of the lung by the bacilli; also, the effusion keeps the parts at rest, prevents friction, and seems to be to some extent bactericidal. Pinquet believes that the **exudate should not be removed** unless pressure symptoms demand its removal, and then only small amounts should be taken away.

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[1] Deut. Arch. f. klin. Med., Aug. 18, 1899.

[2] Semaine méd., No. 33, 1899.

[3] Brit. Med. Jour., Jan. 20, 1900.

[4] West. Med. Rev., 1899.

[5] Med. Rec., Feb. 17, 1899.

[6] V Congres français de méd. interne, 1899.

[7] Thèse de Lyon, 1899.

Lereboullet[1] reports upon the use of a **serum prepared** by Gimbert **from cow's milk.** He notes that the serum is difficult to prepare, and gives an extended description of the method of preparing it. He states, however, that it has been found useful in all cases of debility and depraved nutrition. It is particularly valuable in phthisis. It is also said to be a good vehicle for the administration of medicines.

E. Maragliano[2] discusses the **serum treatment** of tuberculosis. He has produced an antitoxin which seemed to cause some degree of immunity in human beings, and when injected with tubercle toxin in healthy animals, prevented the toxic effects of the toxin. The antitoxin was harmless; it had no direct influence upon the bacilli, acting purely upon the toxin. In this way it probably caused a modification of the soil and interfered with the activity of the bacilli, perhaps entirely destroying them finally. C. P. Ambler[3] describes his results from the use of serotherapy in the treatment of consumption. One hundred cases treated within a year are reported. He found that patients thus managed did not relapse so readily as when other treatment is used, and there are fewer bad symptoms. In his experience the most satisfactory serum has been Fisch's antiphthisic serum, T R. He begins with the injection of 0.2 cc. and gradually increases it to 1 cc. or as much as 2 cc. He has not found the antistreptococcic serum of much value in mixed infection. His general results were: apparently cured, 41 cases; much improved, 31; improved, 14; stationary, 11; died, 2. A. M. Holmes[4] describes 50 cases of tuberculosis in which he used antitoxin. Nineteen incipient cases showed improvement in every instance, and only 1 case relapsed. Of 31 more advanced cases, in which bacilli were found in the sputum, 4 were cured and 5 improved markedly; of 20 cases that were decidedly advanced, 4 improved decidedly, 7 slightly and subsequently became worse, 3 were unimproved, and 6 died.

A. Petruschky,[5] in discussing the **specific treatment** of tuberculosis as carried out in Koch's institute, states that the general results have been unsuccessful in cases complicated with secondary infections, and sometimes these cases grew worse. In discussing the possible results of the specific treatment with tuberculin, he mentions the following: lack of result because of improper dosage, lack of immunization, a variation from time to time between immunization and overloading with the toxin, and also acute or chronic overintoxication with the toxin. The preparation should be given in very small doses, the increase being regulated by determining which amounts produce decided local effects but very little general reaction. Petruschky thinks that a permanent cure may

[1] Bull. Acad. de méd., July 11, 1899.
[2] Berl. klin. Woch., Dec. 4, 1899.
[3] Jour. Am. Med. Assoc., July 8, 1899.
[4] Jour. Am. Med. Assoc., Oct. 7, 1899.
[5] Berl. klin. Woch., Dec. 18 and 25, 1899.

usually be expected from tuberculin treatment if periodic courses of treatment are undertaken, 2 courses usually being sufficient. In 22 cases which he reports, 4 of which were rather advanced, cure was apparently achieved in all.

J. Bounhiol,[1] in a clinical study of **tuberculin R,** reaches the conclusion that it may cause severe local reaction, disturbance of the temperature, and an unfavorable effect upon the appetite. He has also found that tuberculin R has no good effect upon tuberculosis, either in immunizing or in curing the patients, these results corresponding with those obtained by other French authors in their experimental work on animals.

R. B. Klebs[2] considers the use of **tuberculocidin** superior to that of tuberculin in the treatment of phthisis. He thinks specific treatment is essential. The lack of success is often due to one of two causes —mixed infection or gastric disturbance. He attributes much of the gastric disturbance to atrophy of the thyroid, and has found that fresh juice of thyroid gland caused marked improvement in the gastric symptoms. This was not true of thyreodin. Finding that typhoid fever sometimes caused improvement in tuberculosis, he treated mixed infection in tuberculosis with the enzyme of the typhoid bacillus, with some favorable results.

## RHEUMATISM.

**Etiology and Pathology.**—[We have had occasion in the past to call attention to the probable infectious character of rheumatism. In the light of our present knowledge there can be little doubt of this opinion.] R. Oppenheim and A. Lippmann[3] report their bacteriologic investigation of 10 cases of acute articular rheumatism. In 6 cases they had positive results, discovering in each instance a **diplococcus** of slightly elongated shape, with a capsule which stained by Gram's method. Inoculation into animals caused death in about 36 hours in the guinea-pig, while the rabbit and the monkey showed no results except a slight elevation of temperature. In one case of very severe rheumatism the diplococcus was found in the fluid of an exudative pleurisy. The frequency with which the organism was found led them to conclude that it was of importance in the pathogenesis of the disease.

Melkich[4] reports that in examining the blood of 25 cases of acute rheumatism he found in 21 instances the **bacillus described by Achalme;** in 3 cases staphylococci and streptococci were also observed, and in these there was endocarditis. As the temperature reached normal the bacillus of Achalme disappeared from the blood. Melkich also found the Achalme bacillus in 1 case of malaria.

Westphal, Wasserman, and Walkoff[5] report the case of a girl of 19 who had acute articular rheumatism, and a few weeks later developed severe chorea with marked mental symptoms. At that time there was no involvement of the joints. The heart was rapid, though the sounds were clear. She became worse, the temperature rose, and she died in collapse. Endocarditis was found in the mitral valve and there was acute nephritis, and cultures from the heart valve, the blood, and the brain showed a micro-organism which, upon experimental inoculation into animals, produced fever and multiple arthritis after an incubation period of from 5 to 10 days. The organism was usually a **streptococcus,** though at times it presented itself in the form of a **diplococcus.** It grows only in media of high alkalinity and containing much peptone. The authors do not consider that this organism is necessarily the cause of acute articular rheumatism. They state that this is the first demonstration in a chorea following rheumatism of a micro-organism which produces joint effects. The organisms were found only in small numbers in the cadaver, and it would probably be very difficult to find them during life if they were present.

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[1] Thèse de Lyon, 1898, 1899.

[2] Berl. klin. Woch., Dec. 11, 1899.

[3] Compt. rend. de la Soc. de Biol., Mar. 3, 1900.

[4] Arch. russes. de pathol., Sept. 30, 1899.

[5] Berl. klin. Woch., July 17, 1899.

Kronenberg[1] reports a case in which articular **rheumatism followed an operation upon the nose.** The man had had nasal obstruction for years, and hyperplasias of the inferior turbinates were found. The disease was removed by snare from one side. Three days afterward there was a follicular tonsillitis, which improved. Some time later the other side was operated upon, and a week after this second operation the man developed rheumatism of the knees, and then of the ankles, the right elbow, and the shoulder. There was pericardial friction, and also pleurisy and consolidation of the lung. The man died. Kronenberg believes that the operation on the nose caused the infection and death, and insists that it is very important to have thorough exposure and drainage of the field of operation.

F. A. Packard,[2] after an extended consideration of the literature and of his own experience, decides that the **tonsils act as barriers** to the entrance of micro-organisms, as do other lymphadenoid tissues. During or after tonsillitis complication with typical acute articular rheumatism may appear. He considers rheumatism an infectious disease, dependent upon a variety of bacteria, and the phenomena of the disease to be due to toxin absorption. It is probable that the toxin causing rheumatism may be produced by an attenuated micro-organism, and the frequent entrance of the micro-organism by the throat may explain the occurrence of acute articular rheumatism instead of ordinary septicemia or pyemia. He thinks that the terms rheumatic pleurisy, rheumatic sore throat, and the like, should be used with less freedom. Such conditions should be considered the results of infection, whether there are articular phenomena or not, rather than as latent, aborted, or incomplete forms of a condition produced by an unknown, mysterious, intangible rheumatic poison.

D. McKenzie[3] states that during 6 years' practice in the town of Larkhall he noticed that rheumatism was only moderately common in the main portion of the town, while in one part, called Millheugh, which was built on a slope and in a valley, rheumatism occurred with such continuous frequency that it amounted to an endemic. The portion of the town in which it occurred was not especially damp, there was no opportunity for accumulation of surface or subsoil water, and McKenzie thinks that this observation is added **evidence of the infectiousness of the disease.**

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[1] Münch. med. Woch., July 4, 1899.
[2] Phila. Med. Jour., April 21, 1900.
[3] Brit. Med. Jour., May 19, 1900.

J. Bauer[1] describes a case of **acute hemorrhagic polymyositis** which occurred in a man of 39. The symptoms began with severe pain in the muscles of the legs, swelling of the calves, and enlargement of the liver and spleen; the heart became weak and enlargement of the superficial glands occurred. There was but slight fever at any time. Death occurred about 9 weeks after the onset of the disease. Autopsy showed enlargement of the spleen and liver, acute nephritis, degenerative changes, and interstitial hemorrhages in the muscle-fibers, with round-cell infiltration. Pure cultures of **Staphylococcus pyogenes aureus** were obtained.

L. Michaelis[2] gives a preliminary communication concerning the results of his investigation of increase in the **excretion of ammonia** and its cause. It has been shown that the ammonia is usually increased directly in proportion to the increase of destruction of albumin, but in some cases it increases relatively to a greater degree than the general nitrogen excretion, particularly in fevers. Likewise in excess of acids in the system the ammonia combines with the acids and is excreted in excess. In some diseases of the liver there is increased excretion, and artificial oxygen starvation also increases ammonia excretion. Michaelis observed increase in rheumatism, as has been previously noted. He doubted the active relation of the rheumatism, because there was cardiac involvement and distinct circulatory failure. He also observed increase in a case of nephritis with circulatory failure, but ammonia was absent in rheumatism or nephritis without circulatory failure; hence he decided that its presence was due to the disturbance of circulation. He has repeatedly observed it in cases of pure cardiac failure. He thought that it was probably due to the dyspnea, and investigated cases of pure bronchial asthma, finding that in these cases also there was increase of the ammonia excretion, as well as in several cases of infarct of the lung and in a severe case of croupous pneumonia.

**Symptomatology.**—W. C. Bosanquet[3] discusses a report of 450 cases of rheumatic fever admitted to the Charing Cross Hospital within 8 years. They were almost equally divided between the two sexes. Almost all the cases occurred in the second and third decads. In about one-fourth of the cases there was a history of rheumatism in the family, and nearly one-third occurred in domestic servants. It is suggested that the frequency of anemia in the second and third decads may have something to do with the frequency of rheumatism in the same period. The months which produced the greatest number of cases were May and November. Sore throat was the first symptom in about 5% of the cases, and in a small number the disease began with an acute pneumonia. In 69% the knee-joint was affected; the ankle-joint was involved in 47% and the shoulder in 26%. None of the cases were fatal as a result of the rheumatism alone.

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[1] Deut. Arch. f. klin. Med., vol. LXVI, Festschrift.

[2] Deut. med. Woch., April 26, 1900.

[3] Lancet, June 2, 1900.

A. G. Cipriani[1] discusses 3 cases of pronounced **coccygodynia.** He considers this symptom-complex much more frequent than it is usually believed to be or than is indicated by the literature. His cases occurred in a man of 37 and in women of 31 and 40 respectively. They had attacks of extreme pain in the coccyx, which was increased by movements and became excruciating upon defecation. Otherwise the man showed no abnormalities, while both women showed only slight indications of hysteria. The disease in the man was considered to be the result of a chronic arthritis of the spinal column. In one woman the first attack had occurred with an acute articular rheumatism; in the second woman the first attack came on after influenza, while she was pregnant. The pains were controlled by salophen, while other medication was useless, and the author believes that the cases were rheumatic.

Touche[2] describes a case which he uses as a basis of contention for the statement that **tarsalgia** or painful pes plano-valgus may have a rheumatic origin. The patient whose case is described was a 13-year-old child, who had been chilled when very tired. The tarsal joint swelled, there was marked pain, and pes plano-valgus developed. This was followed by the occurrence of a general chronic rheumatism, which led Touche to decide that the affection was rheumatic from the beginning.

**Complications.**—H. R. C. Newman[3] reports a case of rheumatism in which a temperature of 107.8° F. was observed and the hyperpyrexia continued for 24 hours, when the temperature was reduced, and subsequently remained below 102° F. The patient recovered.

J. L. Salinger[4] reports a case of **rheumatic fever with peculiar features.** The patient had probably been sick for 6 days when admitted. He had typical articular rheumatism associated with pericarditis and pleurisy. After his symptoms had improved greatly and the pericarditis and pleurisy had disappeared, and 18 days after the attack had begun, erysipelas appeared and ran a typical course. It was complicated by otitis media. Salinger believes that the rheumatism was ordinary articular rheumatism, and that the erysipelas was of the usual form, due to Streptococcus erysipelatis. He does not consider that the two were due to a common cause.

Weber[5] describes the case of a man who had subacute rheumatism, and who frequently had **albuminuria** and exacerbations of his joint conditions in which fever occurred. Weber thinks that the albuminuria, the fever, and the joint symptoms were all due to rheumatism.

F. P. Morgan[6] records an interesting case of **cerebral rheumatism** which occurred in a man of 29. The man had shortly before had tonsillitis. He showed the usual severe symptoms of rheumatism. About a month after the onset of the rheumatism, without any hyperpyrexia, he developed grave mental disturbance, imagining that he was in the Orient, showing great distrust of the physicians and nurses, and having other delusions of persecution, followed by increasing mental activity, until he became rather violent. There were great restlessness and pain, and on 3 occasions actual convulsions. Morgan particularly directs attention to the fact that cerebral rheumatism is not necessarily associated with hyperpyrexia, and that the cerebral symptoms are not due to the hyperpyrexia alone. No pathologic changes are, as a rule, found in the brain or its membranes after death in these cases. The symptoms are almost undoubtedly caused by toxemia. The diagnosis is usually easy, but one must consider the possibility of salicylate poisoning. The prognosis is grave, more than half the cases proving fatal. The treatment should be symptomatic.

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[1] Il Morgagni, 1899, No. 12.

[2] Gaz. des hôp., 1899, No. 31.

[3] Lancet, June 16, 1900.

[4] Phila. Med. Jour., Nov. 18, 1899.

[5] Edinb. Med. Jour., Jan., 1900.

[6] Phila. Med. Jour., Jan. 13, 1900.

M. Jastrowitz [1] describes 2 cases, both occurring in girls about 20, in which acute rheumatism complicated by chorea was **associated with marked psychic changes.** The first patient had no history of a family tendency to mental disturbance, while in the second case there was a pronounced neurotic history. In both cases there were severe choreiform movements, together with rheumatic involvement of various joints. In both there was much mental excitement with marked change in the intelligence. The first patient had apparently not improved notably when the report was prepared. The second seemed to have become entirely well. Similar cases are referred to, and Jastrowitz states his conviction that the psychoses were due to the direct action of the rheumatic toxin upon the cortical cells—a conclusion that does not seem to be wholly justified, as it seems probable that any severe disease, infectious or otherwise, might have produced the outbreaks in these patients.

**Treatment.**—W. Ewart,[2] in a general discussion of the treatment of rheumatism, states that alkalies should always be used with the salicylates, and that he prefers the citrate of potassium to the carbonate, because the latter is likely greatly to overdistend the stomach by the production of carbonic acid gas. In using a milk diet he has found it advantageous in rheumatism, as in many other conditions, to add about 15 grains of salt to the half pint. He has often observed that milk with salt is readily borne when otherwise it could not be taken.

H. Dayton,[3] as a result of his observation of cases in the New York Hospital, considers the **local application of methyl-salicylate** in acute or chronic rheumatism, or muscular rheumatism, very valuable. The hot-air bath has been found useful in both acute and chronic forms. The use of sodium bromid was very effective in controlling the mental excitement produced by intoxication with salicylate. [We have found the external use of methyl-salicylate highly serviceable.]

Freyberger [4] recommends the **external application of salicylic acid and turpentine** in rheumatism. Neither glycerin nor vaselin should be used in external applications, since they interfere with the action of the salicylic acid. The method which he recommends for making up an ointment is to use salicylic acid, turpentine, and lanolin in equal parts, and to have them put up in lard, the ointment then to be spread on muslin, and the plaster to be tightly applied to the limb by means of a bandage.

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[1] Deut. med. Woch., Aug. 17 and 24, 1899.
[2] Brit. Med. Jour., Mar. 17, 1900.
[3] Med. Rec., April 7, 1900.
[4] Treatment, Nov. 9, 1899.

H. H. Heffernan [1] describes a case of **salicylic poisoning** which occurred after taking 150 grains of the drug at one time. The man had the usual aural symptoms, followed by active and rather violent delirium, with great restlessness, sweating, and a measles-like eruption. The temperature remained normal. The urine was very acid, but contained no albumin or sugar. The man gradually improved, entirely recovering his health in about 2 weeks.

Friedeberg [2] reports some results from the use of **aspirin.** This is acetyl salicylic acid, and occurs in the form of white crystals, easily soluble in alcohol, but with difficulty soluble in water. It has a rather pleasant, slightly acid taste, and is said to have no unfavorable effects upon the stomach, because it is not broken up in the gastric juices, but passes the stomach unchanged. It is likewise stated that it does not affect the heart nor produce deafness, ringing in the ears, or skin eruptions. He describes good results from its use for acute rheumatism, neuralgia, sciatica, migraine, and pleural effusion, and no bad effects were seen. It is not, however, of any special value in chronic rheumatism or in gout. H. Liesau [3] reports, in all, a series of 80 cases in which he has used aspirin with entire satisfaction. In 2 cases it caused some ringing in the ears, but these cases were very susceptible to other salicylic preparations, and practically could not take them. The aspirin was found to have an extremely marked diaphoretic, antineuralgic, and antipyretic effect. It was very satisfactory in both acute and chronic rheumatism, in torticollis, in sciatica, and in exudative pleurisy and pericarditis.

Cagliardi [4] describes 3 cases of acute rheumatism in which he used **lycetol,** a combination of acetic acid and dimethylpiperazin. In one of the cases there was chronic rheumatism, which had been present for 14 years. After about 2 weeks' administration of lycetol in doses of 2 grams a day there was marked improvement in the pains and joint swellings, together with profuse diuresis. The urine at the same time showed a large deposit of urates. The general health improved greatly, and the joints reached almost their normal condition. The two other cases also showed striking improvement.

## DIATHETIC DISEASES.

### GLYCOSURIA.

B. R. Bleiweis,[5] in investigating a number of cases of **infectious diseases** concerning the presence of alimentary glycosuria e saccharo, found it in 9 instances. The cases investigated were chiefly pneumonia, typhoid fever, and erysipelas. The dose of glucose was 100 gm. Three of the cases showed an excretion of between 6 gm. and 8 gm. There was evidently marked difference in the power of assimilation of sugar in the different cases. Alimentary glycosuria was most frequent in pneumonia at about the period of crisis, and was most marked at that time. In 2 cases which showed great reduction of assimilative power he gave starch test-meals, but produced no glycosuria.

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[1] Brit. Med. Jour., April 6, 1900.

[2] Centralbl. f. innere Med., April 14, 1900.

[3] Deut. med. Woch., May 24, 1900.

[4] Boll. delle Clin., 1899, No. 2.

[5] Centralbl. f. innere Med., Jan. 1, 1900.

Hibbard and Morrissey [1] decide from the examination of a large series of cases of **diphtheria** that a transitory glycosuria is seen very frequently in severe cases. It may last as long as several weeks, and is frequently accompanied by albuminuria. Glycosuria was nearly always present in fatal cases.

F. Raphäel [2] reports the excretion of as much as 0.4% of sugar after moderate **atropin-poisoning** in a man who readily developed alimentary glycosuria. The sugar excreted was dextrose. The amount excreted on the first day was 1.8 gm. The glycosuria following the poisoning was not alimentary glycosuria, as the atropin was taken on an empty stomach. The use of atropin in large doses in 5 guinea-pigs caused glycosuria in 4, which in some cases was not alimentary glycosuria, and evidently was due to the atropin. In others it was probably alimentary, as they were given glucose.

K. Morishima [3] found that both **curarin and protocurarin** caused glycosuria inconstantly, the two drugs acting in about the same manner. The amount of glycogen in the liver and muscles had no relation to the degree of glycosuria. In the early part of the poisoning there was marked oliguria, or even anuria, which was followed by polyuria, the amount of glycosuria not being directly related to the polyuria. Glycosuria appeared even when the animals were kept in the cold, but was not seen when the animals were poisoned for the second or third time, even though they had excreted sugar when first poisoned. Morishima used frogs in his experiments.

G. Gobbi [4] has produced glycosuria by the administration of **diuretin.** When administered with glucose, the amount of the glycosuria was much more marked than that produced by the glucose alone. The result was obtained more easily in persons with diseases of the liver and kidneys, and Gobbi considers the glycosuria analogous to alimentary glycosuria. He thinks that the diuretin has a special action on the renal epithelium, facilitating the elimination of sugar when it is present to excess in the blood and tissues. He does not think that the glycosuria was of hepatic origin, though perhaps impaired functionation of the liver increased the tendency to glycosuria.

T. R. Brown [5] reports a case in which **glycosuria** occurred **after operation,** and mentions 2 other cases in which the same condition had been observed. The glycosuria was considered toxic, the result of etherization. He also describes an interesting case termed *malingering melituria*, in which a diagnosis of diabetes had been made by a physician, who repeatedly found sugar in the urine. The patient was extremely neurotic, and it was suspected that the sugar was introduced into the urine. It was found that this was the case, and when her supply of sugar was completely stopped, the sugar disappeared permanently. She used cane-sugar, and the reaction in the urine was considered due to the contamination of the sugar with glucose or to acidity of the urine converting the cane-sugar into dextrose.

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[1] Jour. of Exp. Med., vol. IV, 1899.

[2] Deut. med. Woch., July 13, 1899.

[3] Arch. f. exper. Path. u. Pharmacol., Bd. XVII, S. 28.

[4] Il Policlinico, 1900, p. 159.

[5] Johns Hopkins Hosp. Bull., May, 1900.

G. Hoppe-Seyler[1] states that he has repeatedly observed **glycosuria in the vagrant classes.** This he attributes to insufficient nourishment and to exposure, and consequent disturbance of metabolism.

## DIABETES.

**Etiology and Pathology.**—Töpfer[2] reports that by **injecting the contents of the small intestine** of diabetic subjects subcutaneously into rabbits he produced a glycosuria which persisted for months, and which was largely controlled by dieting. In another series of experiments he made injections into cats, with the same results. The animals did not exhibit either autophagia or polydipsia. In discussion Hammerschlag stated that he has isolated from the intestinal contents of diabetics a bacillus the cultures of which, when injected subcutaneously, provoked a persistent and marked glycosuria. [These experiments must be accepted with reserve. There is as yet no evidence of infection in this disease.]

H. Leo[3] thought that diabetes might be the result of the **action of some toxic substance.** He therefore administered the urine of diabetic subjects to dogs, and was able to cause glycosuria repeatedly by giving the urine by the mouth, though the results were inconstant. He afterward freed the urine of urea and administered it intraperitoneally. Experimenting upon 12 dogs with the urine of 6 diabetic subjects, he found that the urine from 3 cases which were mild produced no results, but urine from 3 severe cases always caused glycosuria, 2% to 3% of sugar being found in the urine.

A. Mathieu and L. Larrier[4] have experimented on the production of **diabetes in animals,** and from the results and the observation of cases decide that glycosuria may result from overproduction of glucose, from insufficient metabolism, or from insufficiency of the kidneys. They found that diabetes produced by complete extirpation of the pancreas is entirely analogous to that seen in human beings, but that pancreatic disease is certainly not always the cause in man, since disease of the pancreas is often not found in diabetes. Also, in animals it is necessary to destroy the pancreas entirely to produce diabetes, and in man it is rarely found entirely destroyed; therefore besides disease of the pancreas, other factors seem to be necessary to the production of the disease. These are reduction in the general vitality of the tissues or of the glycolytic function of the cells. They consider the diabetes of the obese chiefly a nutritive disturbance, while diabetes in thin subjects is often due largely to pancreatic disease. They describe several stages in the diabetes of fat subjects: The first stage is improper metabolism of the carbohydrates, with the production of fat; in the second stage some of the sugar occasionally does not undergo such metabolism, and is excreted in the urine; finally, glycosuria becomes permanent because of the great reduction of the glycolytic function of the cells. In this period the patients may lose their fat and even become much emaciated. Pancreatic and nutritional diabetes are often combined in varying degrees.

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[1] Münch. med. Woch., April 17, 1900.

[2] Verein de Aerztes in Wien., 1899.

[3] Deut. med. Woch., Oct. 28, 1899.

[4] Gaz. des Hôp., 1899, No. 129.

R. Lépine [1] considers that the **glycolytic function of the pancreas** is definitely shown by the following experiments which he has carried out: He took 3 bottles containing sugar, water, and yeast. Into one he introduced a piece of pancreas; into another, a piece of pancreas the nerves of which had been irritated with the faradic current; the third bottle was used as a control. After some hours he observed that the second bottle contained less sugar than the first, the first less than the third. He also noticed that after irritation of the nerves of the pancreas with the faradic current the gland contained more peptone than before; he therefore thought that the peptone excites or increases the glycolytic activity of the pancreas, and suggests that it would be a useful treatment to give nontoxic peptone subcutaneously in diabetes. Such peptones are, however, difficult to secure, and he has not been able to make use of this treatment.

Pregliatti [2] reports, as a result of his experiments on dogs, that after total extirpation of the pancreas alimentary glycosuria did not immediately appear, and he also states that the use of pancreatic extract gave no positive results in these animals. Pregliatti thinks that the probable cause of the glycosuria after the removal of the pancreas is chiefly in **abnormalities of the liver.** He considers that besides the pancreas there is no other organ which has any notable glycolytic action; but the extirpation of the pancreas causes a severe injury to the celiac plexus, and this injury, with the resulting disturbances of the liver function, he considers answerable for the diabetes.

A. Gilbert and P. Lereboullett [3] report 2 cases of **hypertrophic alcoholic cirrhosis** of the liver associated **with diabetes.** They believe that the diabetes was dependent upon the disease of the liver, basing this belief chiefly upon the fact that the diabetes followed the course of the liver disease. In one case proper diet overcame the symptoms of disturbance of the liver, and the glycosuria disappeared. In the other case the cirrhosis progressively increased, and death finally occurred in coma, the sugar persisting in the urine throughout the course of the disease. They also mention a case of tuberculosis with symptoms of diabetes and with enlargement of the liver. This case ended fatally; autopsy showed marked hypertrophy of the liver, without any evidences of fatty degeneration or cirrhosis, and there were no pancreatic changes.

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[1] Lyon méd., 1899, No. 16.

[2] Gaz. degli Osped. a delle Clin., 1899, No. 88.

[3] Gaz. hebdom. de méd. et de chir., May 20, 1900.

They attribute the diabetes in this case to a pure hypertrophy of the liver with excessive functionation of this organ, and believe that the liver changes were analogous to the changes in organs that occur in acromegaly.

H. Sachs [1] investigated the effect of various forms of sugar in normal frogs, and in others whose livers had been removed. He found that extirpation of the liver caused marked reduction of the tolerance for levulose, but did not affect the tolerance for grape-sugar, galactose, or arabinose. Disease of the liver in human subjects also seemed to reduce the tolerance for levulose, but not for the other sugars. He notes that levulose, among several sugars used, is the one best borne by diabetics; and since disease of the liver seemed to affect the tolerance for levulose only, he decides that these experiments are added proof that the **liver is of little importance** in the causation of diabetes.

P. F. Richter,[2] in discussing the question of **renal diabetes,** states that but one case of diabetes—that of Kolisch and Buberl—has ever been reported in which it seemed proved that the glycosuria was due to abnormal permeability of the kidneys. Richter produced glycosuria by intravenous injections of corrosive sublimate, but he attributed this to the action of the liver, since the amount of sugar in the blood was abnormally high. He found, however, that minute doses of cantharadin (0.0005 gm.) caused glycosuria with only an extremely slight increase in the sugar of the blood. The kidneys showed decided changes, chiefly in the vessels of the capsules and in the other capillaries, and Richter thinks that there is some probability that the glycosuria in this case was due to changes in the kidneys, though he does not consider this at all definitely proved.

Eger [3] describes 2 cases in which there was a prolonged history of **nephritis followed by** symptoms of **diabetes.** He considers that they are probably instances of renal diabetes, but admits that he can not substantiate such a statement by any observations of the amount of sugar in the blood. It is, of course, possible that there was a hyperglycemia, and in such case there would be no evidences of the renal origin of the diabetes.

H. D. Beyea [4] contributes an interesting report of the occurrence of **diabetes in a case of multilocular pseudomucinous cystadenoma** of the right ovary. The woman was about to be operated upon when it was discovered that she had sugar in the urine and the general symptoms of diabetes. Operation was undertaken, nevertheless, because of the gravity of her condition. A cyst weighing 22 pounds was removed. Subsequent to the operation the diabetic symptoms grew less, and after about 6 months had entirely disappeared. Beyea suggests that, because the contents of the cyst consisted chiefly of the glycoproteid pseudomucin, it seemed possible that there was some absorption of this substance with excretion of the carbohydrate portion of the sugar and the general symptoms of diabetes. However, there have been other cases of disease of the female generative organs in which there were diabetic symptoms, and in some instances these improved or disappeared after operation which removed the local trouble. There was no apparent opportunity for the cyst to have interfered with the function of the pancreas or the liver in Beyea's case.

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[1] Zeit. f. klin. Med., Bd. XXXVIII, Hefte 1, 2, u. 3.

[2] Deut. med. Woch., 1899, No. 51.

[3] Deut. med. Woch., No. 51, 1899.

[4] Phila. Med. Jour., Jan. 27, 1900.

W. Sternberg [1] reports some experimental work concerning the **origin of diabetic coma.** By intravenous injections of β amidobutyric acid he produced typical symptoms of diabetic coma, with the curious deep respirations characteristic of this form of coma. The α and γ amidobutyric acids produced no such effect. It seems probable to Sternberg, from this and previous work, that the β amidobutyric acid is the cause of diabetic coma, particularly since acids of the β series are found in this condition. He speaks reservedly, but suggests that further study of β amidobutyric acid should be carried out, and that the methods of reducing it or neutralizing it should be investigated with a view to the proper treatment of diabetic coma.

D. Gerhardt and W. Schlesinger [2] describe some interesting and important results from a study of the **excretion of calcium, ammonium, and magnesium** in diabetes mellitus. They placed a normal person and a diabetic on a meat and fat diet. They found that this diet caused the appearance of acetone, diacetic acid, and β oxybutyric acid in both subjects, as well as in themselves when they took the same diet exclusively. To the 2 persons investigated they gave for a series of days 20 gm. of sodium bicarbonate; they then omitted the alkali for a series of days, and subsequently gave it again. During the alkali days the excretion of calcium in both subjects was much reduced and the excretion of ammonia was about parallel; while when the alkali was withdrawn, the excretion of calcium and ammonia in both increased greatly; in the diabetic subject there was decided calcium loss as compared with the amount which was taken in the diet. In the diabetic the use of alkali caused a decided relative retention of nitrogen and apparently prevented a proteid loss. They also found that in the diabetic case when no alkali was given the excretion of calcium in the urine was about 67 % of the whole excretion, while normally only from 10 % to 30 % is found in the urine. The abnormalities in calcium excretion are attributed to the fact that in acid intoxication the organic acids unite readily with the calcium, and thus carry off an excessive amount of it; this also results in the excretion of a greater portion of it through the urine. There was apparently a relative retention of magnesium.

Waldvogel [3] also investigated the **origin of acetone** by administering to fasting persons a diet of pure proteids and a carbohydrate diet. In neither case was acetone formed; the carbohydrates, in his opinion, as in that of many others, seemed to reduce the amount of acetone. Hence fats seem to be the source of acetone. [This opinion is coming to be quite generally accepted.] He also found that in starving persons acetonuria could be produced or increased by administering fats by the mouth, but not when they were given subcutaneously; hence some alterations occurring in the fats in the gastro-intestinal tract seemed to be responsible for the origin of the acetone.

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[1] Zeit. f. klin. Med., Bd. XXXVIII, Hefte 1, 2, u. 3.

[2] Arch. f. exper. Path. u. Pharmacol., vol. XXII, No. 1.

[3] Centralbl. f. innere Med., July 15, 1899.

H. G. Geelmuyden[1] states that besides causing glycosuria **phloridzin** gives rise to decided **acetonuria** in starving dogs. This was slight if they were given proteids or carbohydrates, particularly if carbohydrates were given, but it always occurred if the dose of phloridzin was large. He could not determine to what extent the use of fat in the food influenced the production of acetone, but the administration of butyric acid by the mouth in doses of about 2 gm. caused decided increase in the acetonuria; if given hypodermically, the butyric acid had no effect.

H. Lüthje,[2] in investigating the origin of acetone, administered strychnin to dogs for the purpose of causing convulsions. It was not found in these dogs, however, nor in human subjects after epileptic convulsions. Also, there was **no evidence of its production in the intestine** through bacterial changes, since the free administration of calomel had no influence upon the excretion of acetone.

F. Voit,[3] in investigating the **amount of acetone** excreted **in the urine and by respiration** in a dog on various diets, found that when the amount of meat was large there was a decided increase of acetone, which was not influenced by giving fats or starches. Starvation diminished the amount of acetone decidedly. The acetone in the expired air was always greater in amount than that in the urine, contrary to conditions found in human beings. The quantity, however, was not large; still, he considers it possible that in diabetics the amount excreted in this way may be of importance.

E. Wienland[4] investigated the changes occurring in milk-sugar in the body, particularly in the intestine, in order to determine **whether the milk-sugar** after passing the intestinal wall **produces glycogen.** He found in all the mammalian animals he examined a ferment in the intestine which acts upon milk-sugar, and which he terms lactase. In rabbits he was unable to cause any accumulation of glycogen in the liver by using milk-sugar. There was a marked increase in the respiratory quotient. This he thinks was due to the action of the bacteria in splitting up the milk-sugar; when milk-sugar was mixed with the intestinal contents of the rabbit and digested, 38 % of it was found after 24 hours to have disappeared, the greater part having been changed into acids. He believes that one is not justified in thinking that milk-sugar is transformed into dextrose or galactose in the intestinal canal of the rabbit. When the milk-sugar was given subcutaneously, there was practically no change observed in it.

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[1] Zeit. f. physiol. Chem., Bd. XXVI, p. 385.

[2] Centralbl. f. innere Med., No. 38, 1899.

[3] Deut. Arch f. klin. Med., vol. LXVI, Festschrift.

[4] Zeit. f. Biol., Bd. XXXVIII, 1899.

C. R. Cohn,[1] in considering the question of the **production of sugar from albumins,** investigated the possibility of the formation of sugar from leucin, which closely resembles grape-sugar chemically. He gave leucin to starved animals, estimated the amount of glycogen in the liver, and found that after the use of leucin there was much more glycogen present than in control animals.

**Symptomatology.**—K. Grube [2] reports upon the **condition of the knee-jerk** in 32 cases of diabetes. In the mild forms it was absent in about one-half, in the severe cases in one-fourth, the cases. In 11 cases there was a bilateral neuritis, in 2 of which the cause was possibly alcohol; in the others it seemed to be due to the diabetes. Variations in the degree of glycosuria had no evident effect upon the neuritis; Grube thinks that usually sugar is present in considerable quantity when a diabetic neuritis develops, but after it has developed the amount of sugar has little influence upon it. Hyperglycemia may produce either acute painful irritation of the nerves, neuritis, or a slow degeneration of the nerves; the latter most commonly affects the crural nerve and causes loss of the knee-jerks.

Nararre [3] describes a case of diabetes in a woman of 67 in which a severe increase in the symptoms **followed a wasp's sting.** Nararre thinks that it is probable that the poison of the wasp caused the increase in the symptoms, though he admits that the fright might readily have caused this.

L. Rankin [4] reports, under the heading of **phosphatic diabetes,** the case of a physician who had uric acid calculi, and later developed attacks of profound sleepiness, associated with the formation of a scum on the urine, which proved to be phosphates. He lost in health almost continuously, and finally died in coma after an attack of renal calculus. At one time he drank large quantities of buttermilk, and during this period the phosphates disappeared from the urine, probably because the acid of the buttermilk combined with the excess of phosphates. Rankin believes that in treating phosphaturia we should use much larger quantities of acid than are usually recommended.

E. J. Blackett [5] reports his observation of the occurrence of typical symptoms of **diabetes mellitus** in a case that had presented symptoms of **syphilis.** The patient was a man of 54, and signs of diabetes and syphilis appeared after severe exertion. Apparently all the symptoms of diabetes mellitus were present except glycosuria, and subsequently, after further exertion, he became suddenly prostrated, developed coma, and died after 36 hours. At this time he had glycosuria.

J. H. Sequira [6] describes the case of a child with diabetes in which the diabetic symptoms, including the glycosuria, disappeared almost completely during an attack of jaundice. The child lived, however, only a short time afterward.

H. L. Elsner [7] states that he has observed but 3 cases of **acute**

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[1] Zeit. f. physiol. Chem., Bd. XXVIII, p. 211, 1899.
[2] Lancet, July 22, 1899.
[3] Lyon méd., 1899, No. 47.
[4] Lancet, Mar. 24, 1900.
[5] Lancet, Nov. 25, 1899.
[6] Lancet, July 15, 1899.
[7] Phila. Med. Jour., Aug. 19, 1899.

**diabetes,** all of which occurred in children and, after a rapid course, ended in coma. In one of these cases there was a history of injury of the head, but in no other was there any family history of importance. The other case occurred in a man, the diabetes coming on after severe exposure. Partial coma developed within 3 days, and the man died a week after the onset of his illness. During the last day of his life catheterization was undertaken every 2 hours throughout the day, and about a quart of urine was removed each time. Elsner mentions a case of apoplexy in a syphilitic and alcoholic subject in which glycosuria occurred shortly before death.

A. F. McKenzie[1] reports a case of acute diabetes which occurred in a young man of 20, the diabetic symptoms first appearing one month before death, and the end coming on with coma. [There was, however, no observation of the patient in the preceding period, and diabetes might have been present during this time.]

E. L. Lees[2] describes a case of **acute diabetes in a boy** of 5. The child had apparently been well until 10 days before admission to the hospital, when the symptoms of diabetes appeared. When admitted to the hospital, he had severe thirst, marked glycosuria, and was very much prostrated. He went into coma the next morning and died during that day. There was no history of heredity in the case. The rapid onset of the coma was considered to be possibly due to the excitement attendant upon the removal of the child to the hospital.

T. B. Futcher[3] reports a case of diabetes in a man of 35 in which **lipemia** occurred. The diabetes had developed rather recently, but showed severe symptoms. The blood showed large numbers of refractile granules which had Brownian movement and resembled bacteria. After it was centrifugated the serum appeared turbid and milky, and at a later date was found to contain a considerable number of droplets which took a faint stain with osmic acid and also showed some reaction to Sudan III. Many of them, however, took only a yellowish stain. The lipemia disappeared later, the diabetes became much less marked, and the granules of the blood were present in about the normal number.

**Complications.**—C. S. Wallace,[4] in investigating the records of St. Thomas' Hospital, found 26 cases of **diabetic gangrene** recorded in 11 years. Wallace thinks that usually in cases of gangrene the presence of glycosuria is rather an indication for operation than a contraindication, and considers that the limb should be removed early before severe sepsis has occurred. He thinks that there is no evidence that gangrene occurs in diabetic subjects unless there is associated arterial disease, which itself is likely to produce gangrene. Examinations of the arteries in 24 of the 26 cases showed marked atheroma in all but one instance. The patients were all in advanced life, and there was a very large preponderance of males. Operation was done in 11 cases, with 7 deaths; of 13 cases treated by palliative measures, 8 ended in death and the other 5 were unrelieved.

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[1] Canad. Pract. and Rev., Nov., 1899.

[2] Jour. Am. Med. Assoc., Oct. 21, 1899.

[3] Lancet, Dec. 23, 1899, p. 1735.

[4] Lancet, Dec. 23, 1899.

R. Laudenheimer,[1] in considering the **mental disturbances** which occur in diabetes, states that he does not believe that it is proved that progressive paralysis of the insane can be caused by diabetes. There is, in rare instances, a combination of symptoms similar to paralytic dementia which seems directly due to the diabetes, and which is often greatly improved by proper diet; but any real semblances of progressive paralysis are extremely rare, having been found but 5 times in 1412 cases. Laudenheimer describes 3 cases of diabetes in which there were mental disturbances resembling progressive paralysis of the insane, but in 2 cases there was marked alcoholism, and other complicating factors were present. In the third case the symptoms were those of paralytic dementia, and the diabetes alone seemed to be active in causing it.

C. O. Hawthorne [2] reports an instance of **paralysis of the external rectus muscle** in a case of diabetes mellitus which was thought to be due to peripheral neuritis, because the paralysis of this muscle was complete but isolated. There was also in this case a condition of one retina similar to that seen in chronic nephritis, the retina showing white spots with stellate lines radiating from the macula. There were no evidences of nephritis. The other retina was normal.

P. Ranaut [3] describes the case of a man who had shown the first signs of **acromegaly** at the age of 21, and this had progressively increased until the disease had become typical. He had an attack of intense headache, with disturbance of sight, particularly in the left eye, and with this attack an onset of excessive appetite and polyuria. The urine was examined at once, and contained sugar. The polyuria became as great as 20 liters per day, and he passed as much as 1200 gm. of sugar in a day. The diabetes became of most intense degree within 6½ months, and there were several attacks of coma, which were overcome by the use of alkaline injections. The man finally died of a right-sided pneumonia. Autopsy showed in place of the pituitary body a lobulated soft tumor of reddish-brown color. Histologically, it showed partly the structure of the pituitary body, but in other parts it was evidently sarcomatous. The liver and spleen were greatly enlarged, the kidney still more so, and the pancreas and thyroid gland, the heart, the stomach, and the intestine were all enlarged; the appendix had a length of 13 cm. There was, in other words, a general visceral gigantism, the bones not showing marked enlargement, probably because the disease had developed so late in life.

O. Leichtenstern [4] discusses **diabetic laryngitis.** In some cases of diabetes he found that the first symptom was dryness of the larynx and pharynx, which was not associated with increased thirst or polyuria, and in several instances had been treated merely as catarrh of the larynx. He has also seen diabetic furunculosis of the larynx. The fruity odor of the breath which was associated with the furunculosis led to examination of the urine, and showed the presence of a large amount of sugar. The characteristics of diabetic furunculosis of the larynx are an acute onset of edema and rapid abscess formation, the process occurring in different parts of the larynx. The abscesses improve rapidly after evacuation and are not accompanied by marked constitutional disturbance. The perichondrium was not affected in the cases which he saw. He considers the condition analogous to the furunculosis of the skin which occurs in diabetes.

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[1] Arch. f. Psych., Bd. XXIX, Hefte 2.
[2] Lancet, Sept. 30, 1899.
[3] Gaz. des Hôp., Mar. 23, 1900.
[4] Münch. med. Woch., April 17 and 24, 1900.

H. Eulenstein,[1] in discussing the **diseases of the ear in diabetes,** states that there is no testimony that inflammations of the internal ear are due directly to this disease. As to the middle ear, he reports a collection of 50 cases, 4 of which he describes for the first time, in which there was otitis media in diabetes. Many of these cases might be readily attributed to causes such as influenza, angina, etc. In some, however, a distinct gangrenous necrosis of the ossicles occurred, and in these the diabetes seemed to be the chief factor, though probably it acted in many cases merely by making the patient more susceptible to the occurrence of ear disease.

F. Nagelschmidt[2] discusses the relation between **psoriasis and glycosuria.** Psoriasis does not occur commonly in diabetes, but diabetes seems rather common in patients who have psoriasis. The cases of 25 patients with psoriasis were investigated as to the presence of alimentary glycosuria, using 100 gm. of glucose. Glycosuria was found in 8 after this dosage. In 3 of these the glycosuria might have been due to other conditions, but in 5 it seemed to be related to the psoriasis only. Other skin affections were investigated with negative results.

**Diagnosis.**—A. Katz,[3] in discussing the importance of alterations in the amount and character of the fat in the feces, describes his own work upon this question, which, together with a study of the literature, has led him to the conclusion that if 70% or more of the fat is neutral fat, this speaks strongly for a partial or complete **loss of pancreatic action.** In acute disease of the pancreas the disturbance of the fat-splitting action is much more marked than in chronic cases. The sign is of little importance in sucklings and in cases with profuse diarrhea, since sucklings have normally but little pancreatic activity, and when there is diarrhea the fat-splitting ferment has no time to act. He considers, however, that in diabetes and in any cases which show icterus, if there is a decided reduction of the fat-splitting action, there is probably involvement of the pancreas.

F. A. Larne[4] stated that he had found sugar in urine of a specific gravity as low as 1002.

P. Mayer,[5] noting that some authors consider that morphin appears in the urine in combination with **glycuronic acid,** examined the urine in a case of morphinism, and, besides acetone and diacetic acid, found a substance which, after boiling for some time, suddenly reduced Fehling's solution. Such a reaction occurs with paired glycuronic acids and pentoses. The urine did not ferment. It was probable that the substance was glycuronic acid, since the movement was levorotatory, while pentoses rotate to the right or not at all; and also, after isolating the substances by adding dilute acid, heating caused diminution of the levorotation, and finally caused the urine to rotate to the right, apparently because the paired glycuronic acid was converted into free glycuronic acid. Glycuronic acid has decided reducing powers and rotates polarized light to the right when not paired. When paired, the glycuronic acid compounds, while still reducing copper solutions, rotate to the left.

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[1] Deut. Arch. f. klin. Med., vol. LXVI, 1899, Festschrift.

[2] Berl. klin. Woch., Jan. 8, 1900.

[3] Wien. klin. Woch., 1899.

[4] Jour. Am. Med. Assoc., Aug. 26, 1899.

[5] Berl. klin. Woch., July 3, 1899.

I. H. Corait,[1] in investigating the **usefulness of the phenylhydrazin test** for glucose, used it on solutions containing lactose, glucose, levulose, maltose, and saccharose, and found that all these gave some crystal formation, the crystals being of various sizes and shapes. Glycogen and starch did not react. Substances normally found in the urine also gave no reaction. Glucose reacted in solutions as weak as 1 : 10,000. The presence of albumin had no influence either in preventing reaction or in causing it.

J. A. Grober[2] describes a **refractometer** made by Zeiss which is introduced directly into fluids, and permits of the calculation of the amount of refractive substance contained therein by obtaining the refraction coefficient. His special purpose is to call attention to the value of this instrument in directly calculating the amount of sugar in the urine. He has controlled the accuracy of the refractometer by studying normal solutions with it, and finds it very exact.

A. R. Elliott[3] describes a **new method of using copper** for the detection of sugar in the urine. It is said to be more delicate than other methods, and the reagents are more stable. One solution contains copper sulphate, 27 grains; glycerin, 3 drams; distilled water, 2½ drams; liquor potassæ up to 4 ounces. The second is a saturated solution of pure tartaric acid in distilled water. A dram of the copper solution is heated in the test-tube to the boiling-point, 3 drops of the second solution are added, the mixture is boiled again, and then the suspected urine is added drop by drop.

Rosin[4] describes a method of determining the reducing power of urine, blood, and other body-fluids. He uses **methylene-blue as an indicator,** making a solution of 1 : 3000. In working on urine 5 cc. diluted 5 times with water are placed in an Ehrlenmeyer flask and 1 cc. of liquor potassæ is added; the fluid is covered with liquid paraffin and carefully heated almost to boiling, taking care that air is not allowed to come in contact with the fluid; then 1 cc. of the methylene-blue solution is added by introducing beneath the layer of paraffin a pipet containing it; the mixture is then heated and the blue color disappears; while still hot, a 1 : 100 normal solution of permanganate of potassium is added until the blue color returns. The amount of oxygen in the permanganate solution used indicates the reducing power of the urine.

C. Oppenheimer[5] reports a **new test for acetonuria.** Dilute

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[1] Boston M. and S. Jour., Nov. 23, 1899.

[2] Centralbl. f. innere Med., Feb. 24, 1900.

[3] Kingston Med. Quart., Jan., 1900.

[4] Münch. med. Woch., Oct. 24, 1899.

[5] Berl. klin. Woch., Sept. 18, 1899.

200 cc. of concentrated sulphuric acid by adding a liter of water; to this add 50 gm. of yellow oxid of mercury and stand the solution aside for 24 hours. In the test one adds to 3 cc. of unfiltered urine a few drops of the reagent. If albumin is present, precipitation occurs at once; if not, the precipitate is seen some time later. More reagent is added until the precipitate becomes permanent. The mixture is set aside for a short time, filtered, about 2 cc. more of the reagent are added to the filtrate and 3 cc. or 4 cc. of 30% sulphuric acid. The mixture is then heated in boiling water. If a white precipitate occurs, this indicates acetonuria. If only a small quantity of acetone is present, a large amount of the reagent must be used; and if sufficient of the sulphuric acid is not added, there is some danger of the reduction of the mercury by the heating without the presence of acetone. Such errors may easily be avoided, however, and the test is said to be very delicate. It reacts to diacetic acid, and if bile is present, it reacts by giving a green color to the filtrate. The test may be made quantitative by using definitely measured quantities of urine, adding an excess of the reagent and water, and then heating it on a water-bath. The precipitate is collected, washed thoroughly, dried, and weighed; multiplying the weight by 0.055 shows the amount of acetone present.

**Treatment (Diet).**—F. W. Pavy [1] discusses the differentiation of the **two forms of diabetes** which he has previously described—the **alimentary** and the **composite**; the former being the variety in which he believes the system is unable to assimilate the carbohydrates properly, and the composite being that in which the proteid materials are broken down and the sugar contained within them is set free in the circulation, thus leading to glycosuria. Pavy considers that sugar, when taken into the system, should be partly converted into fat and in part united with nitrogenous substances to form proteids. In the alimentary form of the disease the conversion into fat is interfered with; in the composite form the synthesis into proteids is imperfect. The chief dividing-line is the ferric chlorid test; if this becomes positive, it indicates that the case has assumed the composite character. A combined observation of the urine and of the body-weight indicates the proper amount of carbohydrates to be used. If the carbohydrates are withheld, no loss of body-weight occurs if there is no toleration for carbohydrates, and the patient may even gain weight; if there is toleration for carbohydrates, and they are withheld, the weight falls. He therefore believes that if the urine is free from sugar under restricted diet, and there is reduction in weight, it indicates a favorable prognosis. In the alimentary form of the disease there is no tendency to diabetic coma, this complication occurring only in the composite form. The alimentary form is mild, and its chief danger lies in its tendency to assume the composite character. If in the treatment of the alimentary form the diet has been restricted, the urine becomes free from sugar and the patient begins to lose weight; this indicates that he has acquired some tolerance for a larger amount of carbohydrates, and that this form of food should be increased. Pavy considers diabetic coma to be probably due to $\beta$ amidobutyric acid, as Sternberg believes.

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[1] Lancet, June 23 and 30, 1900.

R. Kolisch,[1] in discussing the dietetic treatment of diabetes, insists that diabetes is **due to excessive production of sugar.** He thinks that the proteids stimulate this excessive production, and that a diet composed exclusively, or almost exclusively, of meats and fats is harmful. In grave cases he recommends purely **vegetable diet.** He thinks that fewer vegetable heat calories than animal heat calories are necessary to maintain life. The vegetables do not stimulate the nutritive functions, and more carbohydrates can be used in this way than when the diet is largely composed of meats. He also recommends a milk diet in many cases.

G. Rosenfeld [2] investigated the readiness with which **dextrose, galactose, and mannose** appeared in the urine of dogs, and found that 50% or more of galactose and mannose was excreted in the urine, the galactose appearing somewhat more readily. All but about 20% of the dextrose was assimilated. Of the corresponding alcohols,—dulcite, mannite, and sorbite,—it was found that dulcite and mannite appeared readily in the urine, sorbite much less readily. Comparative tests were made of galactose and pentacetylgalactose; galactose appeared more readily in the urine than the pentacetyl preparation, but the latter did not cause an increase in the glycogen of the liver. It seemed probable, therefore, that it was not entirely broken up into acetic acid and galactose, but underwent some other form of reduction. Mannite, when given to diabetic cases, behaved as does dextrose, the limit of tolerance being about the same as that for dextrose. Rosenfeld retains the divisions of diabetic cases into mild and severe, but makes a subdivision which he considers important. For instance, each of two mild cases can take 100 gm. of sugar without glycosuria. It will often be found that one will assimilate large quantities of added amounts, while the other excretes all over this amount. The first case would be much more favorable in prognosis. Also of severe cases it will be observed that certain of these excrete sugar even when carbohydrates are not given. Some will assimilate a portion of the carbohydrates that may be given, while others will excrete the whole amount. The same **difference in prognosis** applies to these cases as to mild cases.

Von Jaksch [3] has investigated the effects of **arabinose, xylose, and rhamnose** in diabetes. The first caused a marked increase in nitrogen metabolism, usually upset the bowels, and was in larger part excreted itself; the second caused an increase in the excretion of sugar, though it was not excreted itself; rhamnose was excreted in considerable quantities itself, the amount of glucose excreted was not decreased, and diarrhea was caused. Hence v. Jaksch decides that these substances can not be used as substitutes for glucose in the treatment of diabetes.

Mosse [4] recommends the **use of potatoes** in place of bread in dieting diabetics. He has found that when potatoes are substituted for bread, the glycosuria is likely to diminish and the patients to improve in general condition; while if bread is used again, the more severe symptoms return. R. Saundby,[1] in discussing the treatment of diabetes, also recommends that potatoes be used more freely than they are instead of bread; the bulk is larger for the same quantity of carbohydrates, and they are more satisfying to the patient and better borne. The best form of alcohol he considers to be Scotch whisky. It is almost free from sugar.

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[1] Wien. klin. Woch., Dec. 28, 1899.

[2] Centralbl. f. innere Med., Feb. 17, 1900.

[3] Deut. Arch. f. klin. Med., Aug. 18, 1899.

[4] V Congres français de méd. interne, 1899.

W. Winternitz and A. Strasser[2] report their results from the use of **absolute milk diet** in diabetes mellitus. They state that usually it reduced the sugar strikingly or caused it to disappear entirely within 48 hours in a number of subjects that had been resistant to treatment; and in others, in whom the diabetes had followed obesity, injury, or shock, the results from absolute milk diet were excellent. If albuminuria were present, it usually disappeared; also, acetone often decreased in amount if present, and appeared if it had not been present. If the cases relapsed after the milk diet was stopped, the use of this diet the second time caused the disappearance of the sugar again. In one case sugar did not return for 5 months while under observation. Usually the body-weight decreased at first, but then increased. The authors claim that the ability to assimilate other forms of carbohydrates is no criterion of the ability to assimilate grape-sugar.

Masuyama and Schild[3] discuss the treatment of diabetic steatorrhea with **preparations of pancreas.** They state that it has been demonstrated that removal of the pancreas or occlusion of the pancreatic duct causes great diminution in the absorption of fats, with relatively little effect upon the fat-splitting action. [This statement may be criticized at once as being directly against the most satisfactory work upon the question.] They report their results in the case of a diabetic who had lost much weight, and who passed about 75 gm. of sugar a day. The feces were fatty. The fat absorption was determined throughout 4 periods. In the first it was found that only 36.9 % was absorbed; in this period no pancreas was given. In the second 100 gm. of scraped fresh pancreas were given each day, the fat in the food being kept at the same point; during this period the fat absorption was 63.8 %. In the third period the pancreas was excluded again, and the absorption was about the same as before. In the fourth period the pancreatic juice obtained by compressing the pancreas under high pressure was used in doses of 100 gm. to 125 gm. per day; during this time 45.51 % of fat was absorbed. Hence fresh pancreatic substance seemed to aid decidedly in the absorption of fats, and was more useful than pancreatic juice. The patient objected to the use of fresh pancreas after it had been taken for a time.

Burghart[4] has used **didymin** in treating diabetes, because the male sex is likely to be affected, and the subjects are likely to be obese and to suffer from impotence. Burghart believes that he had good results from this treatment in older men, while it was valueless in young subjects.

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[1] Lancet, May 19, 1900.

[2] Centralbl. f. innere Med., Nov. 17, 1899.

[3] Zeit. f. diät. u. phys. Therap., 1899, vol. III, p. 481.

[4] Deut. med. Woch., Sept. 21, 1899.

T. Oliver [1] gives a subsequent report of a case previously described in which apparent recovery from diabetic coma had followed upon the use of **saline transfusion.** He states that the patient remained well for 243 days, when there was another onset of coma, and death occurred within 2 hours.

G. D. Barney [2] has repeatedly had successful results from the use of the **double bromid of gold and arsenic** in the treatment of diabetes mellitus. The subjective symptoms grow less marked and the glycosuria decreases. He gives 5 drops 3 times a day, increasing a drop a day.

## GOUT.

**Etiology and Pathology.**—C. Watson [3] has carried on some investigations of metabolism in gout, particularly determining the **phosphate metabolism,** and at the same time the changes in the **character of the white blood-corpuscles.** The point of interest is that the myelocytes were found in the blood, both during the interval and during the attacks, but were present in much larger number during the attack. Also, he observed a decided retention of phosphates during the first 3 days of the attack, and after this a decided increase; he thinks that there may be some relation between the changes in the excretion of phosphates and the presence of myelocytes, and suggests that one should especially investigate the condition of the bone-marrow in gout. He found the uric acid excretion to be decidedly increased during the first 3 days of the attack, the total nitrogen being increased at the same time, and increasing more decidedly later, while the uric acid excretion dropped somewhat. Watson does not think that the alkalinity of the blood is diminished during the attack. He thinks that the excretion of uric acid is increased rather than diminished, and that the amount of uric acid in the blood is not greater during the attack than in the interval. He insists that there is no evidence that uric acid is the direct cause of the acute attack, and agrees with others in his statement that uric acid has been too exclusively studied in investigating gout. The use of sweetbreads caused some increase in the excretion of phosphates; potassium iodid and sodium salicylate had somewhat the same effect.

W. Bain,[4] after investigating the effects of **excretion** of **phosphorus, uric acid,** and **alloxur bases** in gout when the patient is on a fixed diet, decides that the excretion of phosphorus varies directly with that of uric acid, and that the xanthin bases are not increased. Sodium salicylate increased the excretion of uric acid; the same is observed after taking thymus gland. As to the determination of the amount of uric acid in the blood, Bain considers that the methods of investigation are not sufficiently delicate to permit of accurate determination of moderate changes.

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[1] Lancet, Aug. 26, 1899.

[2] N. Y. Med. Jour., Mar. 31, 1900.

[3] Brit. Med. Jour., Jan. 6, 1900.

[4] Brit. Med. Jour., Oct. 28, 1899.

W. His, Jr.,[1] discusses the **excretion of uric acid** in gouty persons during the attack and in the interval. The average quantity excreted by gouty persons either in the interval or during the attack is about the same as that in normal persons. He finds, however, that there is always in the early stage of an acute attack of gout a decided reduction in the amount of uric acid excreted, and sometimes its entire disappearance. This usually occurs on the day before the attack, sometimes as long as 3 days before. He considers that it is probably due to the deposition of uric acid from the blood about the joints, and that the subsequent increased elimination is due to the excretion of the deposit of uric acid, chiefly through the action of the leukocytes. The effect of drugs upon the elimination of uric acid was investigated. No marked effect was seen from alkalies or vegetable acids; lithium seemed to decrease the elimination slightly; piperazin, lysidin, urocedin, and urotropin were without effect. Colchicum caused a decided increase.

Badt [2] reports his results from the estimation of uric acid in 5 cases of gout, estimations being made during the attack and subsequently. He found that the **uric acid was not lessened** in any instance during the attack, but was rather greater, though the increase was so slight that he lays no weight upon it. There was no decrease in excretion of urine.

J. F. Goodhart,[3] in speaking of gout and the origin of this disease under the term of acidity, states that the belief that uric acid produces the disease is due merely to the eagerness of the profession to grasp something tangible in developing theories concerning any disease. With practically all others who have worked upon the question, he considers that varying amounts of uric acid are merely evidences of differences in metabolism or in food. **Uric acid itself does not produce disease.** If there is a tendency to excrete an excess of uric acid, he believes that this will continue even after the diet is confined to foods which do not contain uric-acid-forming elements. He thinks that the diet in gout should be liberal, and should be limited only in special details made necessary by the individual case. The diet should, in particular, not be monotonous, as this is likely to arouse disgust and to reduce the nutrition of the patient. Large quantities of water should be given for the purpose of flushing out the general tissues and the urinary tract especially, in view of the possibility of causing some solution of calculi.

Caro [4] describes a case of **epilepsy** in which he investigated the **excretion of uric acid** daily for about 2 weeks. During this time 2 severe attacks occurred, and each time on the day preceding there was a very great reduction in the excretion of uric acid. [This is of only minor interest, however, as these results can not be trusted, because there was apparently no attention paid to the diet taken during this time, and, furthermore, the method used is antiquated and unreliable.]

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[1] Deut. Arch. f. klin. Med., Sept. 29, 1899.

[2] Zeit. f. klin. Med., Bd. XXXVII, Hefte 5 u. 6.

[3] Lancet, Jan. 6, 1900.

[4] Deut. med. Woch., May 10, 1900.

C. F. Martin[1] discusses Kolisch's **theory that gout is due to xanthin bases.** Kolisch believes that these bases are increased in gout, because, he thinks, the kidneys are diseased and the xanthin bases are found in excess, since the kidneys are unable to convert the nucleic acid derivatives into uric acid in sufficient amounts. Kolisch believed that he demonstrated such an increase in nephritis, but the method used by him (Krüger-Wulff) was wholly untrustworthy. Martin has used Salkowski's method in determining the amount of xanthin bases present in a number of cases of nephritis. In only 1 case was there any large increase in the xanthin bases, and in this case only one determination could be made. In the others the amount excreted was entirely within the normal range. There was, however, in no case anything approaching a predominance of the nitrogen of the xanthin bases over that of the uric acid, a condition which Kolisch considered to be present, and there was certainly no evidence of any marked change in the xanthin bases. In several cases Martin found the percentage of uric acid high, entirely contrary to Kolisch's reports that the uric acid is diminished in disease of the kidneys. He notes that in 2 cases of intermittent albuminuria the urine after resting, and when almost free from albumin, showed much less uric acid than after exercise, when albumin was present. Hence he judged, probably justly, that the kidneys were in less capable condition after exercise than after resting, and that nevertheless more uric acid was excreted after exercise, an evidence that damage to the kidneys does not decrease the uric acid excretion.

J. Weiss,[2] in investigating the production of uric acid, administered croton oil, because this drug produces marked irritation in the intestinal mucous membrane and causes a leukocytosis. He felt that the result might be an increase in the uric acid, but no such effect was observed. He did see a marked increase in uric acid after taking thymus gland or pancreas, but this increase did not occur if quinic acid were given at the same time.

A. E. Taylor[3] reports some very important results from his investigations in his own person of the **influence of various diets upon** the elimination of **nitrogen, uric acid, urea,** and **xanthin bases.** He put himself on varying diets for 3 days before beginning his estimations, and then with each diet determined the amount of the substances mentioned present in the 24 hours' urine for 6 successive days. In the first period he used a normal mixed diet, excluding foods containing much nuclein; he also used the same diet with coffee and beer in order to increase the nucleins; likewise a diet composed chiefly of sweetbreads, one almost exclusively composed of beefsteak, a vegetable diet, a milk diet, and one consisting of sago, butter, and sugar, and containing almost no nitrogen. He found that with the normal diet and the milk diet the uric acid was lowest; it was very largely increased with the vegetable diet, and was excessive during the sweetbread diet. The use of coffee and beef together with the normal diet largely increased the uric acid; large amounts of meat did not increase the excretion of uric acid. The excretion of xanthin bases bore, in general, the same relation to the nitrogen as did the uric acid. The fact demonstrated positively by these experiments was that, as has previously been determined but is now made more evident, the excretion of uric acid varies directly with the amount of nucleinic compounds taken in the food by healthy persons. Haig's gout ratio was found to be present under conditions very different from those described by Haig. Wholly contrary to this author's teaching, the gout ratio was present when no proteid was taken; and the more meat ingested, the further did the ratio differ from the so-called gout ratio. The work on the xanthin bases seemed to confirm that of Krüger and Salomon, who found that only about one-third of these bodies is produced by metabolism, the remaining two-thirds being derived from the foods. As a result of these experiments it may be stated very positively that investigations of the elimination of uric acid and the purin bases, and, on the other hand, of the total nitrogen, must be carried out separately, since the amount eliminated is evidently entirely distinct, and in both cases dependent chiefly upon the character of the food rather than upon any varying conditions in the individual.

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[1] Phila. Med. Jour., Dec. 23, 1899. [2] Zeit. f. physiol. Chem., Bd. XXVII, S. 216.
[3] Am. Jour. Med. Sci., Aug., 1899.

H. Kronka [1] investigated the **effect of meat diet** on the production of **gout in hens.** After they had been fed on meat for from 3 to 5 months symptoms of gout appeared. The hens showed an unsteady gait, which he attributed to pain in the legs, swelling of the joints, debility, with paroxysmal increase of the symptoms. In the more acute cases there were marked deposits of urates, and in marked chronic cases there were distinct tophi formed, and deposits of urates were regularly found in the kidneys. The elimination of uric acid was decidedly increased, but this elimination decreased when lime was administered.

A. C. Croftan, [2] in a critical review of theories concerning uric acid in gout, states that he considers it demonstrated that uric acid is not a product of the oxidation of albumin, but of nucleins, and that the chief disturbance of nuclein metabolism is an increase of the xanthin bases with a decrease in the formation of uric acid, which is the theory promulgated by Kolisch. He considers it demonstrated that the alloxuric bases are highly toxic, and he states that his own investigations have shown that when xanthin bases or hypoxanthin are injected in a 3% to 7% solution into animals, granular degeneration of the epithelial cells lining the tubuli contorti and proliferation of the endothelium of the intratubular capillaries may be seen. Albumin is always found after a period of 3 weeks. [It may be fairly said that this is the only testimony that xanthin and hypoxanthin are poisonous when taken in reasonable amounts. The author does not mention the actual quantity which he used, but it is reasonable to consider that the quantity was larger than the amount found in the daily urine of human subjects, and hence these results can not be considered to be of much value. It is certain that there is no evidence that the xanthin bases are increased in gout or have any direct relation to the causation of gout.] Croftan recommends that inhalations of oxygen be used in the treatment of the gouty diathesis in order to increase the oxidation of nucleins.

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[1] Berl. klin. Woch., Jan. 1, 1900.

[2] Jour. Am. Med. Assoc., July 8, 1899.

**The Local Lesions.**—W. His, Jr.,[1] reports the work which he has undertaken with his assistants concerning the **causes** and **effects of the local lesions** in gout. The injection of suspensions of acid sodium urate caused symptoms like those seen in gout and similar local changes. There was a cellular infiltration and a subsequent necrosis, the necrosis extending beyond the limits of the deposits of urate crystals. It seemed evident to His that the uric acid deposit caused the necrosis, and that therefore Ebstein's theory that necrosis is primary is probably incorrect. Subcutaneous injection caused deposits resembling tophi, but injections about the joints did not cause deposits in the cartilages, though they produced symptoms resembling those seen in an attack of gout. The deposit of uric acid was carried away both by phagocytosis and by solution by the tissue-juices. M. Freudweiler's[2] previous work had presented the same results, the **injection of biurate of sodium** causing necrosis with inflammatory infiltration which subsequently formed connective-tissue capsules around the necrotic areas and urate deposits. Control injections of calcium carbonate caused very much less marked changes. The attempts which he made to alter the alkalinity of the blood and thus to influence the absorption of the deposits were without result. Freudweiler injected suspensions of the biurate into his own arm and caused inflammation. Excision of one of the areas and examination of the tissues showed conditions corresponding to those seen in the animals. The use of Faching water, Wiesbaden gout water, and phosphoric acid had no influence upon the deposits.

W. His., Jr.,[3] has repeated Freudweiler's experiment of injecting urate of sodium beneath the skin of the rabbit. He, too, found that a small nodule was formed, which was surrounded by embryonic fibrous tissue. The salt remained amorphous. By feeding the animal with small amounts of alcohol for a considerable time the capsule surrounding the nodule became well-formed connective tissue and the arrangement of the salt became radiate.

A. Ritter[4] investigated some of the chemical **alterations of the urine favorable to** the production of **uric acid sediments.** Mixtures of monosodic and disodic phosphates with acid sodium urate and urea produce varying effects, according to the amount of the different phosphates used. The use of monosodic phosphate in large amounts resulted in the deposition of crystalline uric acid; disodic phosphate either prevented such a deposition or lessened the rapidity with which it occurred, but when large amounts of this phosphate were present, the acid sodium urate was deposited from a solution. He thinks, therefore, that the disodic phosphate is important in the causation of uric acid deposits in acute attacks of gout. He believes that the deposit of urates is primary and the necrosis secondary.

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[1] Verhandl. d. XVII. Congress. f. innere Med., 1899.

[2] Deut. Arch. f. klin. Med., Bd. LXIII, Hefte 3 u. 4.

[3] Deut. Arch. f. klin. Med., Feb. 6, 1900.

[4] Zeit. f. Biol., Bd. XXIV, p. 155.

E. Schreiber[1] believes that the reason attempts to produce **uric acid infarcts** have failed is that the animals that have been experimented upon (chiefly dogs) are able to excrete large amounts of uric acid, and also to oxidize the major portion of the uric acid that is given them and excrete it in another form. Schreiber thinks that the production of infarcts is probably dependent, in the first place, upon damage to the kidney; and, secondly, upon deposition in the damaged areas. He attempted to produce proper conditions for the production of infarcts by administering aloin and corrosive sublimate, but the experiments were negative. He thinks that the frequency of infarcts in very young infants is the result of the fact that they excrete very large quantities of uric acid in the early days of life, and that this damages the very delicate epithelium of the kidneys of such young subjects and thus gives opportunity for deposition of uric acid.

**Symptomatology.**—Moscati[2] reports a case which he considers one of gout because there was a decided hereditary gouty tendency, and the manifestations of disease appeared paroxysmally after dyspeptic attacks and were associated with marked urinary deposits of uric acid. Unusual features present were decided **enlargement of the spleen,** the appearance of marked **glandular swellings** in the peritoneal cavity, and a **temporary pseudophlegmon.** Improvement of the general condition caused the swellings to disappear. He also describes a case of gout in which there was swelling of the thyroid gland, and refers to reports of others in which there appeared lymphatic and tonsillar swellings and pseudophlegmons in gout.

Strumme[3] reports a case of severe gout which finally ended fatally, the symptoms in the latter part of life being chiefly those of nephritis, though there were also slight swelling of the lymph-glands and severe cachexia. The chief point of interest in the case was the postmortem discovery of amyloid degeneration in most of the organs.

J. Dunn[4] describes cases of asthma which improved upon the use of colchicum after resisting other treatment. He therefore attributes them to uric acid collemia.

A. S. Myrtle[5] describes an attack of gout occurring in his own person, which terminated in an acute myositis of the entire left leg. Constant electric baths brought about a cure.

**Treatment.**—W. Bain[6] believes that, while it is not proved that **foods producing uric acid** are damaging to gouty subjects, until further investigation shows that they are not harmful the amount given gouty subjects should be very small. He believes that regulation of the diet according to the condition of the gastro-intestinal tract is the best method. Exercise should be ordered according to results, and severe fatigue should never be produced. He has observed gout in vegetarians, and thinks that meat should be admitted to the diet of the gouty. Sodium salicylate he does not consider useful in gout; it seems to increase the excretion of uric acid at the expense of the leukocytes.

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[1] Zeit. f. klin. Med., Bd. XXXVIII, Hefte 4, 5, u. 6.

[2] Morgagni, 1899.

[3] Deut. Arch. f. klin. Med., Bd. LXIV, Festschrift.

[4] Va. Med. Semi-Monthly, Sept. 22, 1899.

[5] Brit. Med. Jour., June 9, 1900.

[6] Brit. Med. Jour., No. 28, 1899.

W. B. Bain [1] reports upon the **excretion** of uric acid, ammonia, phosphates, and alloxur bases **with mixed diet, vegetable diet,** and **animal diet** in the gouty subject. Uric acid increased with a vegetable diet, while it decreased with a meat diet. Sodium salicylate caused a slight increase in the uric acid and alloxur bases. Guaiacum caused decided increase in the uric acid without any increase in the phosphorus and alloxur bases, hence it probably increased the elimination of uric acid, and should be a valuable prophylactic in gout.

A. Haig [2] in treating headaches has the patients **avoid animal food** except milk and cheese, and excludes tea, coffee, and vegetables which contain large quantities of nuclein. The proteids in general are reduced so that 3 to 3½ grains of urea are excreted for each pound of body-weight; the salicylates are given to further the excretion of uric acid.

A. P. Luff,[3] in considering the **relation of the various forms of sodium biurate** to the production of gout and to the treatment of this disease, states that he has found that sodium bicarbonate rapidly transforms the soluble gelatinous form of the biurate into the insoluble crystalline form. This, he thinks, explains the occurrence of acute attacks in gouty subjects after the use of large amounts of sodium bicarbonate, the latter substance causing deposition of the crystalline biurate. Hence the potassium salts are more valuable in the treatment of gout than sodium salts, because the potassium salts, on the contrary, lessen the rapidity of the conversion of the gelatinous biurate into the insoluble crystalline form. Luff also states that he has found the alkalinity of the blood in gouty persons even higher than in health, the increase of the alkalinity being due to increase in sodium salts. In a further consideration of the causation and treatment of gout Luff [4] states that the onset of an acute attack is due largely to inability of the kidneys to excrete normal amounts of uric acid, so that some accumulation takes place in the blood, the symptoms not being produced by uric acid in solution, but being caused by precipitation of the crystalline forms. In acute attacks of gout he uses colchicum, citrate of potassium, and purgation with blue pill, administering the potassium salts in large amounts and giving large quantities of water. The general treatment of gouty cases depends greatly upon the use of proper diet, which, in Luff's belief, is chiefly the avoidance of a large admixture of proteids and carbohydrates at any one meal. There should also be careful attention to hygiene, and the potassium salts should be freely used.

E. P. Adams [5] believes that the **treatment of gouty diathesis** should be almost **exclusively** a **dietetic** one, since medicinal treatment is of little value. Whether it be due to excessive production or to deficient assimilation, he agrees that there is always an excess of urates in gouty diathesis, and these are increased by the use of nitrogenous foods, alcoholic drinks, exhaustion, or lack of muscular exercise, or by cold and dampness; hence these conditions should be removed as far as possible, and water should be drunk, since it carries uric acid and urates from the body. Coffee he admits in the diet if it has a diuretic effect; if not, it should be definitely excluded. Milk is very useful. Table salt should be taken in limited quantities, as its presence in the lymph near the joints has a tendency, he states, to hasten the formation of the insoluble biurate of soda and its deposition. Leguminous vegetables should be avoided because of the large amount of nitrogen they contain. Corn he recommends, as he does greens, potatoes, beets, tomatoes, and almost all fruits; fish may be well used as a substitute for meat. He also recommends fats, starch, and sugar.

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[1] Brit. Med. Jour., April 7, 1900.

[2] Ibid., Nov. 4, 1899.

[3] Ibid., Oct. 28, 1899.

[4] Lancet, Nov. 18, 1899.

[5] Jour. Am. Med. Assoc., Dec. 23, 1899.

W. Tunnicliffe and Rosenheim,[1] after a careful study of uric acid salts, decided that there is **no evidence that quadriurates occur** in urinary deposits or in the fluids of uratic deposits of the body, and they think that the term quadriurate may well be dropped. The substances obtained under the conditions when quadriurates are said to form really consist, they believe, of mixtures of uric acids and biurates, of pure uric acid, or of pure biurates alone. The variations in the behavior of uric acid and its salts may, they consider, be due to the existence of 2 forms of uric acid.

W. J. Morton [2] recommends the use of **electricity** in rheumatism, dividing this disease into muscular rheumatism, acute and subacute rheumatism, and chronic articular rheumatism. He also states that electricity is useful in gout and rheumatoid arthritis. The result of this treatment of muscular rheumatism is, he says, that the pain is relieved, but returns after about 24 hours; after repeated treatment the pain is permanently relieved. In articular rheumatism he has had better results from the electrostatic treatment than from any other, and gout, he asserts, in the acute attack, often subsides completely. It usually controls the pain entirely at any rate, though it may return. Repeated applications will give permanent relief. Rheumatoid arthritis, he states, may be completely arrested at any stage. The rapidity with which this is effected depends upon the stage to which the disease has advanced.

E. L. McGinnis [3] reports successful results in 2 cases from the **treatment of gouty tophi by cataphoresis.** He wrapped the joints in absorbent cotton, saturated it with iodid of lithium, and applied the galvanic battery.

Giofredi,[4] after finding the internal use of **piperazin** and other medication useless in the treatment of a gouty tophus about the tendon sheath of the peroneus longus, used this drug hypodermically in doses of $\frac{5}{6}$ of a grain, 10 injections being given. Pain followed each injection, but this was decreased in the later injections by previously spraying the parts with ether. The tophus was completely absorbed during this treatment. W. Fearnley [5] has had excellent results in the treatment of gout from the use of piperazin when combined with the use of the Harrogate sulphur waters.

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[1] Lancet, June 16, 1900.

[2] Med. Rec., April 21, 1900.

[3] N. Y. Med. Jour., April 7, 1900.

[4] Gaz. degli Ospidali, Aug. 20, 1899.

[5] Brit. Med. Jour., Dec. 30, 1899.

Witthauer[1] has found **asparin** very useful in the treatment of those cases commonly given sodium salicylate. He has found it particularly valuable in gout, rheumatism, pleurisy, and similar conditions. It was very effectual, and did not disturb the stomach or heart.

Hermann[2] reports satisfactory results from the use of **glycerin** internally **for uric acid calculi.** In 115 cases he noticed in 60% relief of pain or discharge of the calculi. From 1 to 4 ounces of glycerin dissolved in an equal quantity of water were given between meals, and repeated, if necessary, for several days. In some cases he observed headache, and in 3 diarrhea. These symptoms soon disappeared, and there were no other unfavorable results, but it suggested to him that the dose be decreased at first below that mentioned. Albuminuria is not increased. Hermann thinks that the good results were not due to the solvent action upon uric acid, but to the lubricating action upon the epithelium of the urinary tract.

A. Nicolaier,[3] in an extensive experimental study of **urotropin** and its influence upon genito-urinary conditions, notes that the development of micro-organisms is influenced by urotropin only when the urine is kept at about body-temperature. He found that at this temperature formaldehyd is formed from urotropin, and he thinks that this explains the effects of urotropin. The presence of acids, including uric acid, aids in the formation of formaldehyd from urotropin, hence he thinks that the uric acid of the urine is active in setting free the formaldehyd. He finds also that urotropin is a solvent for uric acid, probably chiefly as a result of the presence of the formaldehyd which is formed from the urotropin. Acting upon this belief, he has obtained useful results in the treatment of uric acid gravel by urotropin. He has found that almost all cases of cystitis improve upon the use of urotropin; tuberculous cases alone being uninfluenced, and sometimes even damaged. Cases of pyelitis also almost always showed decided improvement soon after the use of urotropin was instituted. He has found, too, that the uric acid diathesis is favorably influenced by the use of this drug. H. E. D. Brockman[4] recommends the use of urotropin as a urinary antiseptic in any condition of the urinary tract in which antisepsis is necessary; also as a diuretic in some cases; and more particularly as a uric acid solvent in gout, uric acid diathesis, and gravel.

## RHEUMATOID ARTHRITIS.

C. A. Bannatyne[5] reports a case of rheumatoid arthritis in a woman of 57. After the disease had been present for 12 years there occurred an attack of acute colitis with acute arthritis; this was followed by pericarditis; later by pleurisy, first of one side and then of the other; and finally by death from bronchopneumonia. Only one knee-joint could be examined after death; in this there was found acute destructive disease of the joint without any cartilaginous thickening or outgrowth. The thickening of the mucous membranes in this case was thought to be a **secondary infection** and to be distinct from the joint disease.

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[1] Therapist, Aug. 15, 1899.

[2] Med. Chron., Jan., 1900.

[3] Zeit. f. klin. Med., Bd. XXXVIII, Hefte 4, 5, u. 6.

[4] Lancet, June 30, 1900

[5] Brit. Med. Jour., Oct. 14, 1899.

J. S. Billings, Jr.,[1] describes a case in which there were the usual appearances of rheumatoid arthritis, and in which he observed a reduction of the red blood-corpuscles to 1,344,000, the leukocytes giving a count of 9200, and a percentage of lymphocytes of 84.3. There were no nucleated red cells, and Billings thinks that the case was one of **pernicious anemia.** There was a **family history of arthritis deformans** in the mother and maternal grandmother.

**Treatment.**—In the treatment of rheumatoid arthritis R. A. Baylis[2] recommends the constant wearing of woolen garments, residence in a dry climate, attention to the digestive organs, and the use of hot baths with hot and cold douches; massage and hot air are also valuable. Locally, he recommends the use of **guaiacol and salicylate of methyl** in olive oil; he also gives guaiacol bicarbonate internally. When acute inflammatory symptoms are present, he uses hot boric acid dressings; or if the joints become distended, he aspirates them.

G. W. Collinson[3] describes a case of rheumatoid arthritis which occurred in a young woman of 22 and had been present for 11 years, first involving one ankle after an injury, and subsequently extending to most of the other joints with the exception of the small joints of the fingers. The right elbow was completely ankylosed and the left elbow almost entirely so. Collinson first **excised the right elbow-joint,** and afterward the left, with the result that while the patient had previously been unable to feed herself, she was able 6 months afterward to use her arms with fair comfort and ease and to earn her own living.

Taylor[4] recommends the use of **electricity** as a bath in relieving the pains of rheumatoid arthritis.

Bier[5] considers the good results obtained from **hot-air** treatment of joints, owing to the hyperemia produced. He induced hyperemia by an apparatus consisting chiefly of wooden boxes lined inside and out with soluble glass to prevent fire. The heat is obtained by a large alcohol lamp, the air being led into the box by an iron funnel. Bier considers the active hyperemia produced by hot air valuable in rheumatism and similar conditions, but useless in tuberculosis, while the hyperemia induced by ligatures gives good results in tuberculosis. Hyperemia may also be incited by a vacuum by using an apparatus similar to a cupping-glass.

## RHIZOMELIC SPONDYLOSIS.

Leri,[6] in an extensive paper on rhizomelic spondylosis, reports a number of cases, and criticizes the diagnosis in a number of instances which have been reported as belonging to this class. He then describes the **changes found in a fatal case** in Mariè's service. The spinal column formed an almost complete arc of a circle in the dorsal and cervical regions, the course of the upper cervical vertebræ being practically at right angles to that of the lumbar vertebræ. There was some abnormal anterior curvature in the lower lumbar regions. The ligaments were strikingly ossified; almost exclusively, however, on the convexity of the curvatures, the dorsal kyphosis showing ossification of the supraspinous ligaments, while over the lumbar lordosis there was ossification of the anterior portion of the discs. There was marked hypertrophy and almost complete ankylosis of the articular extremities. The explanation of the lesions offered is that the trouble begins as a softening of the articular extremities, with consecutive hypertrophy and ankylosis.

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[1] Med. Rec., Sept. 30, 1899.

[2] Edinb. Med. Jour., Aug., 1899.

[3] Lancet, Nov. 4, 1899.

[4] Clin. Jour., Oct. 11, 1899.

[5] Münch. med. Woch., Nov. 28 and Dec. 5, 1899.

[6] Rev. de méd., Aug. 10 and Sept. 10, 1899.

H. Senator,[1] in discussing **chronic ankylosing spondylitis,** describes the case of a man of 65 who had a history of rheumatism and developed stiffness and kyphosis of the spinal column. He then discusses the nature of these cases. He insists that the disease is not new, as has been claimed by Mariè and some of his students. There are 2 forms of spondylitis deformans known, in one of which the disease is primarily of the intervertebral cartilages; the other begins laterally, particularly about the transverse processes and the ligaments, and is associated with marked hyperostosis and calcification. The nervous symptoms may be due to pressure of the spinal roots, or they may be the result of inflammation of the spinal cord of a rheumatic or gouty origin and entirely independent of the changes in the vertebræ. Senator does not believe that there is any reason for describing different forms of spondylitis according to the portion of the spine affected or the coincident involvement of other joints.

G. Kirchgaesser[2] reports a series of cases of **inflammatory necrosis of the spinal column,** and discusses this disease. A characteristic of the disease is that it tends rapidly to involve the greater part of the spinal column, and to produce almost entire stiffness of this joint, with some curvature and torsion of the spine. Most frequently there is anterior curvature in the upper cervical and lower dorsal regions, and the hip-joints are affected also; sometimes the shoulder-joints and joints of the chest are involved. The patients have a characteristic gait, resembling the waddling of a duck. Pain is not a prominent symptom. The causation is often probably rheumatism, or perhaps congenital predisposition, and at times the infection of the gonococcus seems to have been active.

L. R. Müller[3] describes a case which occurred in a man of 28, in which, after an attack of acute inflammatory rheumatism, there was a gradual increase of **rigidity of the spine** until the whole column was found fairly fixed. There was lordosis in the upper cervical region and a kyphosis below this. The hip-joints were markedly affected.

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[1] Berl. klin. Woch., Nov. 20, 1899.

[2] Münch. med. Woch., Oct. 10 and 17, 1899.

[3] Münch. med. Woch., Oct. 10, 1899.

## OBESITY.

G. Rosenfeld,[1] in discussing the treatment of obesity, states that he believes that his work has probably proved that **fat is not formed from proteids,** fatty degeneration consisting apparently in reduction of the amount of proteid or glycogen in the cells and in the replacement of these by previously formed fat particles coming from other parts of the body. It seems probable that fats are formed entirely from carbohydrates and fats, but almost exclusively from the latter. Fat is oxidized with greater difficulty than any of the varieties of food, and it is usually deposited in the form in which it is taken without any decided change in its character. He believes, therefore, that in the treatment the main point is to reduce the amount of fat; and, after this, the chief point is reduction of the carbohydrates.

In treating obesity v. Hoesslin [2] adheres largely to Ebstein's method of diet. This is almost a purely **protein-fat diet.** In the treatment he also employs hydrotherapy, exercise, and thyroid tablets. He does not advise a decided restriction of fluids. He gives bran bread instead of ordinary bread, and reduces the sugar largely, oftentimes allowing things to be sweetened with saccharin. He advises a cold sponge on arising, a half bath 2 hours later, every other day a sweat bath, and a cool fan douche in the early evening. The thyroidin is given in doses of 1 gm. per day. Regular exercise is insisted upon.

G. M. Debove [3] reports the case of a gouty subject who was exceedingly obese. His weight at one time was about 385 pounds; when he came under treatment it was 330 pounds, and his obesity was producing much disturbance of his general health. He was put on **milk diet,** since he had signs of interstitial nephritis. He took at first 2½ liters of milk per day, and during a month lost 30 pounds; in the second month he took 2 liters, and lost about one-half as much as during the first month; the milk was reduced to 1 liter the following month, and this amount was continued for 4 months. He was then losing relatively little weight on this diet, so he was given green vegetables, solids, and fresh fruit. Within a year he had **lost about 120 pounds,** his general health had greatly improved, his gouty symptoms had disappeared, and while he still had polyuria, there was no longer albuminuria. His heart had been acting very badly before the treatment, but after the treatment had been carried out his cardiac action was practically normal except for occasional intermittence.

A. Loewy and P. F. Richter [4] discuss obesity under **two forms,** one which is more or less **mechanical,** the other depending upon an **anomaly in metabolism** which leads to the accumulation of fat. One apparently important factor in the regulation of fat metabolism is the ovarian secretion. The authors have shown that the removal of the ovaries results in marked lessening of the interchange of gases and a considerable increase in body-weight. This suggests that ovarian extract would be useful in the treatment of obesity. On healthy animals ovarian extract, they found, had practically no effect. This is an instance of what the authors consider the true basis of organotherapy: namely, that organic extracts have practically no effect on normal persons, but are merely methods of substituting what has been lost or can not be produced by the body.

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[1] Berl. klin. Woch., No. 30, 1899.

[2] Münch. med. Woch., Sept. 19, 1899.

[3] Bull. Acad. de méd., Mar. 6, 1900.

[4] Berl. klin. Woch., Dec. 11, 1899.

A. Loewy,[1] in investigating the effect of **ovarian extract** upon metabolism of fats and nitrogen, reached the conclusion that it increased the metabolism, but only at the expense of the nonnitrogenous bodies, no increased elimination of nitrogen occurring.

Burghart [2] describes a case of obesity in a girl of 20 who was imbecile and unable to earn her living. She had never menstruated, and examination showed infantile internal genital organs. Because of this, Burghart used **ovarian tablets,** with the result that the fat decreased largely, her intelligence improved markedly, her muscular strength was much increased, and she finally became capable of earning her own living. Menstruation, however, did not occur.

A. W. Sherman [3] describes good results from the use of **thyroid extract** in obesity. He has never seen bad effects from its use.

C. E. Hirsch [4] reports decided improvement in a case of obesity under the use of **iodothyrin.**

## ADDISON'S DISEASE.

C. Phillips [5] describes a case of Addison's disease with **simple atrophy of the adrenals.** Very few cases of this combination have been reported, and almost none have been carefully studied. The clinical features of the case were of interest because of the appearance of a pigmentation of the skin 14 years before the onset of the severe constitutional symptoms; rheumatoid pains were observed early in the disease, the temperature was irregular and atypical, albumin and sugar were absent from the urine, and there was a pronounced anemia with hypoleukocytosis. [The number of the leukocytes is not stated.] The adrenal bodies at autopsy showed only a simple atrophy of moderate degree, with probably some attempts at compensatory hypertrophy. Phillips describes 13 previously reported cases of apparent simple atrophy of the adrenals with Addison's disease. He considers it very improbable that the quantitatively insignificant changes present in this case could have been the direct cause of the Addison's disease. He thinks it more probable that perversion of function of the adrenal bodies rather than total lack of function caused the affection.

R. A. Fleming and J. Miller [6] report what they consider to be an occurrence of **Addison's disease in a number of members of the same family.** The mother had been abnormally pigmented since her first pregnancy, which had occurred about 8 years before the report. Since this time the pigmentation had increased, particularly in the form of black, mole-like spots. She had shown marked gastro-intestinal disturbance and great debility. Four children, aged respectively 7, 5, 3½, and 2½ years, had shown similar symptoms, decreasing in degree according to the youthfulness of the child. All had gastro-intestinal disturbance with decided pigmentation and the appearance of mole-like spots over the body. The authors are in doubt as to whether the cases were really Addison's disease, and believe that more careful attention to the possibility of familial Addison's disease may demonstrate its existence.

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[1] Berl. klin. Woch., Dec. 11, 1899.

[2] Deut. med. Woch., Sept. 14, 1899.

[3] Jour. Am. Med. Assoc., Mar. 24, 1900.

[4] Med. News, Feb. 24, 1900.

[5] Jour. Exp. Med., Sept., 1899.

[6] Brit. Med. Jour., April 28, 1900.

W. F. Cheney[1] describes a case in which there were the typical symptoms of Addison's disease, and the injection of 2 mg. of **tuberculin caused reaction.** The man had no evidences of tuberculosis elsewhere, and the result of the reaction led Cheney to think that he probably had tuberculosis of the suprarenals, and this strengthened his belief in Addison's disease. Autopsy showed marked caseation and enlargement of both suprarenals. There was secondary involvement of the liver and a healed tuberculosis of the left lung, but no other important changes.

F. Buszard[2] describes a case of Addison's disease in which there was **wide-spread pigmentation** and a peculiar appearance of **leukoderma,** with striking white patches on the face, knees, abdomen, palms of the hands, and ends of the fingers. The skin was deeply pigmented about the white patches, which suggested that the pigment had been heaped up about the spots after having been carried from them. The autopsy in the case showed tuberculosis of the lungs and peritoneum and of other abdominal organs, and almost entire destruction of the suprarenal capsules from tuberculosis.

Vollbracht[3] describes a case of **Addison's disease** in a girl of 15 which was associated **with purpura hæmorrhagica.** The girl had severe purpura, and about a year afterward showed typical signs of Addison's disease, which improved after the use of suprarenal gland. She died subsequently with the signs of meningitis, but the postmortem showed no cerebral changes except hyperemia and edema. The suprarenal glands were almost completely destroyed by tuberculous changes. The author believes that the changes in the suprarenal glands had been going on for a long time without the clinical appearance of Addison's disease, and that the purpura was probably an evidence of the damage to the general system through the disease of the suprarenals.

Ménétrier and Oppenheim[4] report a case of Addison's disease which had subacute evolution without marked signs of Addison's disease, death taking place as the result of an infection from a **pneumococcic angina.** The throat trouble was of benign appearance, and they considered that it would ordinarily have been of good prognosis, but it became fatal owing to the lack of suprarenal function and the consequent susceptibility of the patient. Both suprarenal glands showed caseation.

H. W. Evans[5] describes a case which he terms **Addison's disease**

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[1] Phila. Med. Jour., Mar. 24, 1900.

[2] Lancet, Feb. 17, 1900.

[3] Wien. klin. Woch., No. 28, 1899.

[4] Gaz. des Hôp., Mar. 30, 1900.

[5] Lancet, June 9, 1900.

**following typhoid fever.** It occurred in a girl, and in the fifth week of the disease she was taken with vomiting, the pulse became accelerated, and a pigmentation of the skin developed on the abdomen, which rapidly increased in extent and intensity. The patient died of asthenia in the thirteenth week of the disease. No autopsy was made.

J. M. Finny [1] describes a case in which there were present the signs of malignant disease of the lungs, and in which postmortem showed **sarcoma of the suprarenals** with secondary sarcoma of the lung. The man's complexion had been noted to be especially dark, but there is no mention of any other signs of Addison's disease.

Zaudy [2] describes a case in a man of 46 in which there had been very obscure complaints of pain about the chest and abdomen, with vomiting, dysphagia, abdominal tenderness, and enlargement of the liver. There was no stricture of the esophagus, and the case was considered to be probably one of carcinoma of the stomach, though there were some signs of peritonitis. The postmortem showed no lesions of consequence except caseation of the suprarenal glands and swelling of the lymph-glands. Examination at this time showed the presence of some pigmented spots on the temples and lips. Zaudy believes that it was a case of Addison's disease. He refers to Ebstein's previous report of a similar case, and states that if a patient presents a **symptom-complex resembling peritonitis** and severe emaciation, one is justified, in the absence of other explanatory factors, in deciding that it is Addison's disease, even although other signs of this disease are not present.

E. Enriquez and P. Lereboullet [3] report a case of **general arsenical pigmentation** of the skin which occurred in a man of 47 who had taken arsenic for eczema. The dose had been 15 drops of Fowler's solution, and this had been continued for about 3 months. At this time there were attacks of syncope, with general adynamia; and, with pigmentation present, the case closely resembled Addison's disease, so that the arsenic was continued. The pigmentation and emaciation grew worse at first, but afterward the general symptoms improved, and nothing remained except wide-spread pigmentation, which was most marked about the buttocks. The mucous membrane of the mouth showed some pigmentation. There was some arsenical keratosis of the palms of the hands, but there were no evidences of arsenical neuritis. In the previously reported cases of arsenical pigmentation of the skin, as in this case, the pigment was always seen in darker and lighter areas. It usually comes on 2 or 3 months after the use of the drug is instituted. This patient took only about 4 gm. altogether, but even smaller doses have been known to cause pigmentation. The pigment sometimes disappears after the drug is stopped.

E. Sergent and L. Bernard,[4] who had previously reported a case of rapid death with disease of the suprarenal glands, now describe a **clinical**

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[1] Dublin J. M. Sc., Nov. 1, 1899.

[2] Zeit. f. klin. Med., Bd. XXXVIII, Hefte 4, 5, u. 6.

[3] Gaz. hebdom. de méd. et de chir., 1899, No. 54.

[4] Arch. gén. de méd., July, 1899.

**syndrome** which they attribute to **insufficiency of the suprarenal glands,** and refer to a series of cases in which fatal disease was found associated with changes in the suprarenals and in which there were, nevertheless, no appearances of typical Addison's disease. They believe that when the suprarenal glands are destroyed alone, without affection of the nerves, Addison's disease is not produced, but there results a condition resembling that seen after experimental extirpation of the suprarenal glands, the disease running a course of varying rapidity. If the nerves are involved, Addison's disease results. They direct attention to the medicolegal importance of the cases described, and state that in all cases of sudden death in collapse, in coma, after epileptiform convulsions, or after severe injury or operation one should carefully investigate the condition of the suprarenal capsules.

Burghart [1] describes 2 cases of Addison's disease which were treated by **suprarenal extract,** both patients dying without improvement. One was apparently made worse, the use of the extract being followed by fever, loss of appetite, and prostration.

## DISEASES OF THE THYROID GLAND.

### EXOPHTHALMIC GOITER.

**Etiology and Pathology.**—P. Jaunin [2] believes that the condition produced by **chronic iodism** is indistinguishable from thyroidism and exophthalmic goiter. In his experience chronic iodism occurs almost exclusively in subjects of goiter. In such persons small amounts of iodids often produce an attack indistinguishable from exophthalmic goiter. He also found that patients with chronic iodism have a condition which often resembles tuberculosis, but the symptoms vanish after stopping the iodin and instituting proper hygienic methods of living. The repeated use of iodin again renews the symptoms. As exemplifying his belief concerning the close relationship between chronic iodism and exophthalmic goiter, he mentions the case of a girl who had goiter, with prominence of the eyes, excessive sweating, tremor, and emaciation. Iodin ointment had been used for the goiter, and after this ointment was stopped, the girl improved at once and soon became entirely well. In another case a woman of 52, who presented all the symptoms of Graves' disease except exophthalmos, was discovered to have been using iodin ointment and to have taken potassium iodid. Stopping the use of the iodin caused decided improvement in the symptoms, but she afterward died of heart failure. The patient had had the same series of symptoms previously after taking iodin preparations, and the same had been observed in her mother. G. Gautier [3] likewise thinks that chronic iodism and exophthalmic goiter are practically the same condition. His experience in Geneva has been that people coming from elsewhere are extremely sensitive to iodin after taking the water, and that people in

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[1] Deut. med. Woch., Sept. 14, 1899.

[2] Rev. méd. de la Suisse rom., 1899, No. 5, p. 301.

[3] Ibid., p. 618.

Geneva are frequently subjects of exophthalmic goiter or readily present symptoms of this condition if they are given iodin. Swelling in the thyroid gland is often observed in women, and is frequently combined with some atrophy of the breasts. The secretion of milk is also much decreased by the use of iodin. The use of iodin for abnormalities of the teeth in the people about Geneva has often been noticed to produce attacks resembling exophthalmic goiter. Gautier believes that the **waters of Geneva** are likely to be harmful if they are used for more than a few weeks. He describes a number of cases illustrating the points which he makes, some of them having become severely ill after a protracted stay at Geneva. In one case permanent melancholia occurred; in another, mania. Gautier considers that the thyroid gland regains its normal condition only slowly if its function is once disturbed by the use of iodin.

E. Roos[1] has found that the action of the thyroid gland varies directly in accordance with the **amount of iodin** in the gland. He gave dogs thyroid glands of children, which contain a large amount of iodin, and thyroid glands of dogs which had been given potassium iodid in order to increase the amount of iodin in the gland. The action upon metabolism was in direct relation to the amount of iodin in the gland, and if iodin was nearly absent, there was practically no effect. This is in direct opposition to the theory that there are toxic substances in the gland which combine with the iodin and thereby lose their poisonous properties. Roos found that the amount of iodin in the glands of carnivorous animals was less than that in the glands of herbivora.

R. Tambach,[2] in some investigations concerning the **iodin compounds of the thyroid gland,** was unable to find any free thyroiodin in the gland, nor was this substance set free by the employment of artificial gastric and pancreatic digestion. He did, however, find iodo-albumin compounds which were almost completely soluble in water. Digestion of these yielded iodo-syntonin, iodo-albumoses, and iodo-peptone. By further breaking up these bodies with acids or alkalies he obtained free thyroiodin, but the quantity did not correspond to the amount of iodin-containing substances in the gland. The iodo-albumin compounds contained an amount of iodin representing about 96% of the total quantity in the gland. All of this was precipitated by substances which precipitate albumin. Of the remaining 4%, about half was free and readily soluble in water; the remaining 2% was soluble in water, but firmly combined. He learned that the amount of iodo-albumin compounds in the gland varies largely according to the time of year and to the animals from which the thyroid is taken. Tambach considers the iodo-albumin compounds to be the chief active constituents of the thyroid gland.

Oswald[3] extracted thyroid substance with ammonium sulphate, thus obtaining **two proteid bodies** with very different characteristics. One contained iodin, and seemed to belong to the globulins, and this Oswald called **thyreoglobulin.** This iodin-containing substance caused a decided increase of nitrogen metabolism, and Oswald decided that this was the specific substance of the thyroid gland so far as the thyroid influences metabolism. Another substance was found which belonged to the **nucleoproteids** and contained phosphorus, but was free from iodin. The moist thyroid contained about 10% of thyreoglobulin. The colloid material of the thyroid could be found in the lymphatics leading from the gland, and Oswald believes therefore that the gland furnishes a secretion which enters the blood, and that the influence of the organ is not solely through processes which take place within it. He considers that the secretion acts chiefly in regulating metabolism, and that the symptoms presented in cases in which the thyroid action is more or less completely lost are due to intoxication with the products of intermediate catabolism. Normally he thinks that the thyreoglobulin is broken up after it enters the circulation, and that the iodin is separated and carried back to the thyroid gland. He has used thyreoglobulin in 2 cases of myxedema with improvement in the symptoms.

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[1] Zeit. f. physiol. Chem., Bd. XXVIII, p. 40.

[2] Ziet. f. Biol., Bd. XXXVI, S. 549.

[3] Münch. med. Woch., Aug. 15, 1899.

J. Donath [1] investigated the **urine** of normal persons and of persons with Graves' disease for the **presence of iodin** without success. He found that even after administering iodin and giving iodôthyrin in amounts which contained 1.8 mg. of iodin, the methods used did not demonstrate the presence of iodin in the urine. He believes that the available methods are not sufficiently accurate to show the presence of less than 3 mg. of iodin in the urine.

A. Boinet [2] reports the occurrence of symptoms of **Graves' disease** with decided psychic disturbance after the use of **large quantities of thyroid gland.** The thyroid was first given for general dermatitis; the patient was well, except for his skin affection, until at his own instance he took from 6 to 9 sheep's thyroids daily. In about a week there was great restlessness and delirium, anorexia, delusions of persecution, marked tremor, rapid pulse, and swelling of the thyroid. The symptoms decreased after a few days, but were still present in lesser degree. At the end of about 6 weeks the man had practically recovered, when he began to take thyroid gland secretly once more, and the previous symptoms returned. After brief treatment, however, they disappeared the second time.

D. Baldi [3] considered that if the **thyroid gland destroys a poison** which is normally produced in the body, one might expect that such poison would be found in the blood in considerable quantities if the thyroid were extirpated; hence he injected into thyroidectomized young dogs the blood-serum of animals whose thyroids had been removed. No symptoms of poisoning, however, resulted; on the contrary, the symptoms caused by the thyroidectomy seemed to improve. He considered it possible, nevertheless, that a poison was present, and had caused the production of the toxin, and that hence injection of the blood-serum immunized the animals against thyroidectomy to some extent. There seemed, however, to be no real testimony for this belief, since normal serum produced the same effects. Baldi therefore decides that there is no evidence that the thyroid destroys any toxin which is produced in the body, or that, at any rate, no such toxin gains entrance to the circulation.

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[1] Zeit. f. klin. Med., Bd. XXVIII, p. 169.

[2] Rev. Neurol., July 15, 1899.

[3] Arch. Ital. de Biol., XXXI, 1899, p. 281.

B. B. v. Fenyvessy,[1] in discussing the **influence of extract of thyroid** upon the circulation and respiration, found that the latter was little influenced. The blood pressure of rabbits was lowered, the heart action remaining unchanged and the fall in pressure being due to dilation of the vessels. Similar effects may be observed after the use of the extract of the hypophysis, adrenal extract, or peptones. Hence no definite conclusions can be drawn from these results. Iodothyrin had no direct influence upon the blood pressure or the pulse-rate. There was no evidence that iodothyrin had any neutralizing effect upon the poisons that influence the vagus and the depressor nerve.

J. Katzenstein,[2] in investigating the **importance of the thyroid gland** to the organism, completely removed the thyroid gland of one side in dogs and divided the vascular and connective-tissue connections of the gland on the other side, enveloping the latter gland in various substances, chiefly fish bladders, in order to prevent its becoming adherent once more to the tissues. Of the results in 10 animals which he discusses, 3 were fatal at once, and were therefore excluded from consideration ; of the other 7, 4 animals remained entirely well. He decides, therefore, that consideration of this short series of cases makes it probable that the thyroid gland is not necessary to the life of the animal. He learned that by cutting the nerves leading to the gland complete degeneration of the gland could be produced, which showed 2 stages, the first consisting of degeneration of the epithelial cells with loss of distinction between the two varieties of the cells, and the production of what appeared to be a mass of homogeneous colloid material, in some areas no cells being distinguishable ultimately. In the second stage the epithelial tissues and their products vanished entirely and the gland looked like a mass of cavities with folded masses projecting into it, the whole resembling somewhat the folds of the intestine. Even in extremely advanced stages of degeneration the animals remained in good health, and Katzenstein thinks that practically all of the gland may be lost without disturbance of health. He does not think that other glands act vicariously in the place of the thyroid.

H. J. Vetlesen [3] considers that exophthalmic goiter is dependent upon **changes in the nervous system.** In 4 cases he had the thyroid partly excised. Ultimate permanent recovery occurred in 1 case, temporary improvement in 2 other cases, and in the fourth case improvement until pregnancy occurred, when the symptoms became decidedly worse. He has seen thymus gland act beneficially in some cases, but in others it was entirely useless. He recommends sodium phosphate in the treatment of the disease, and has also seen sulphuric acid do good.

P. Londe,[4] in discussing the etiology of exophthalmic goiter, states that he considers that some cases are dependent upon changes in the nervous system, while others are due directly to the disease of the thyroid gland, thus making two distinct classes of cases.

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[1] Wien. klin. Woch., Feb. 8, 1900.

[2] Deut. med. Woch., Nov. 30, 1899.

[3] Zeit. f. klin. Med., Bd. XXXVIII, Hefte 5, 6.

[4] Rev. Neurol., 1899, p. 788.

A. Pader[1] reports a case in which **hysteria** was associated with exophthalmic goiter. He considers this association of the two affections very common, and believes that in such cases there is a common cause for the two. He believes that it is probable that abnormal functionation of the thyroid gland, with intoxication of the system, produces hysteria, in the same way that alcohol and other poisons give rise to hysteric manifestations.

G. Carter,[2] in discussing the causation of Graves' disease, states that it may be due to disease of the sympathetic nervous system, to toxemic changes in the thyroid gland, or to infection. He suggests that it is very possible that there is a **protozoan infection.** This suggestion is based chiefly upon general reasons, from certain of the symptoms, and from the distribution of the disease, rather than upon any demonstration of such infection.

Dinkler[3] reports his investigation of 2 fatal cases of exophthalmic goiter. In one there was marked **hyperplasia of the thymus gland,** and the thyroid showed practically normal conditions. In the other the thymus was not affected, and the thyroid showed the changes usually found in Graves' disease. This leads Dinkler to believe that the changes in the thyroid in exophthalmic goiter are largely due to the condition of the thymus, the thyroid gland being little affected in those cases where the thymus is markedly hyperplastic. In one case he found changes in the central nervous system by using Marchi's method.

**Symptomatology.**—Popoff[4] describes 2 cases of exophthalmic goiter in which **hemorrhages** from the skin and the mucous membrane occurred, the hemorrhages disappearing coincidently with the improvement of the Graves' disease.

W. Uhthoff[5] describes a case of exophthalmic goiter occurring in a man of 27, in which **ulceration of both corneæ** was observed, both ulcerations perforating. Uhthoff had seen the same complication twice previously, but notes the rarity of it.

A. Homberger,[6] in discussing the question as to whether a specific **insanity** occurs with exophthalmic goiter, reports a series of different forms of mental disturbance with exophthalmic goiter. There were 20 cases of melancholia, 18 of dementia, 6 of progressive paralysis of the insane, and also instances of various other forms of insanity, besides 6 cases of hysteria, 17 of neurasthenia with some mental disturbance, and 6 cases of epilepsy. Homberger thinks that while any form of insanity may occur with exophthalmic goiter, there is no indication that any specific form is seen with this disease.

**Treatment.**—Boisvert[7] used **thymus gland** in the treatment of 2 cases of exophthalmic goiter with improvement; in one recovery occurred within 3 months, except that the thyroid remained somewhat swollen. The other symptoms disappeared.

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[1] Thèse de Paris, 1899.

[2] Edinb. Med. Jour., Oct., 1899.

[3] Münch. med. Woch., May 22, 1900.

[4] Neurol. Centralbl., 1899, No. 22.

[5] Allg. med. Zeit., 1899, No. 37.

[6] Inaug. Diss., Strassburg, 1899.

[7] Rev. méd., 1899.

C. E. Hirsch[1] reports 2 cases of exophthalmic goiter which improved decidedly upon the use of **iodothyrin.**

Burghart,[2] following the suggestion of Ballet and Enriquez, removed the thyroids from dogs and afterward used their blood in the treatment of exophthalmic goiter. He also used **blood from a case of myxedema,** preparing it by taking 200 cc. of blood, mixing it with salt solution, and placing it in the ice-chest for 24 hours after adding chloroform. He filtered this and allowed the filtrate to stand until the clear liquid became blood color. This was used for several weeks in doses of 20 cc. to 55 cc. After 8 injections of this blood the patient showed a decided increase in weight, reduction of the exophthalmos, of the sweating and tremor, and of the heart action, and decided increase in general strength. The improvement after 6 months was very decided; there had been, in all, a gain of 23 pounds. Three cases were treated with the blood of the dogs, or with the dried alcoholic extract of their blood, 2 of them improving decidedly and gaining in weight; the other improved somewhat, but left the hospital soon after the treatment was begun.

O. Lanz[3] thought that in thyroid cachexia a poison might be found in the secretions, and that if used in Graves' disease it might have an antagonistic action upon the poison of this affection, hence he **resected the thyroids of goats and used their milk** in the treatment of exophthalmic goiter in 3 cases. One patient left the hospital 2 weeks after the treatment was begun with her pulse much improved and the goiter decreased in size; she was also sleeping much better. In the second case decided improvement had occurred after 9 weeks' treatment. In the third case the patient seemed decidedly better, though the treatment had been carried out for a week only.

C. L. Minor[4] used large **colonic injections of water** in the treatment of 2 cases of exophthalmic goiter in which there were decided gastro-intestinal symptoms. He believed that the disease in these cases was due to gastro-intestinal autointoxication. Recovery occurred in one case and there was marked improvement in the other, and Minor thinks that the results showed the correctness of his belief concerning the origin of the disease.

C. N. Allan,[5] believing that **bile** influences metabolism, gave this substance by the mouth, by hypodermic injection, and by direct injection into the thyroid in the treatment of Graves' disease. He describes 2 cases in which marked improvement was believed to have occurred. In one of them bile pigments were found to be present only in small quantities in the feces, and this led to the use of bile. The woman complained of marked pain, dyspnea, and rapid heart action after the injections; they caused a good deal of suffering, but she received 48,000 grains of bile within 5 weeks. She thought that she had improved very greatly.

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[1] Med. News, Feb. 24, 1900.

[2] Deut. med. Woch., Sept. 21, 1899.

[3] Cor.-Bl. f. schweiz. Aerzte, Bd. XXIX, No. 23, 1899.

[4] Med. Rec., Dec. 2, 1899.

[5] Lancet, Aug. 26, 1899.

Rehn[1] gives a statistical report of a series of **operations for exophthalmic goiter.** In 177 partial excisions of the gland 57.6% were cured, 26.5% improved, 2.3% unimproved, and there were 13.6% deaths. In 32 resections of the sympathetic 31.1% were cured, 50% improved, 12.5% were unimproved, and death occurred in 9.5%. After ligation of the artery in 14 cases 24% were cured, 50% improved, and the remainder died. Resection is recommended as the best operation. Narcosis should be limited as far as possible.

A. Schiller[2] reports 4 operations for exophthalmic goiter. In one case the right thyroid artery was tied, with decided improvement. The woman had previously been melancholy, but after the operation the mental condition rapidly improved and she ultimately became well. In another case a cyst was excised and the patient, a teacher, rapidly recovered and became able to work. In the third case ligation of the thyroid resulted in some improvement, but the patient died later from heart failure. Strumectomy was done in the fourth case, death occurring on the table through entrance of air into a vein.

J. Donath[3] reports a case of exophthalmic goiter in which **resection of the sympathetic** was carried out. Following the operation the pupil became much contracted, and the corresponding side of the face became very red. The thyroid on that side decreased greatly in size. Resection was done on the other side 6 weeks later. The color, temperature, and perspiration of the two sides then became similar, and the exophthalmos decreased. There was some general improvement, interrupted by periods of depression, but at the end of 16 months the symptoms persisted without much real change.

J. H. Nicoll[4] reports a case of exophthalmic goiter in which the thyroid was **excised under cocain anesthesia.** The result was good and the symptoms were considerably improved.

## RELATION BETWEEN EXOPHTHALMIC GOITER AND MYXEDEMA.

Adami[5] contributes an interesting paper on the etiology and symptomatology of goiter. He decides that ordinary endemic simple goiter is directly related to the water-supply, but no definite constituent of the water has been found to be at fault, and probably the condition is due to microbic action. One may trace a **series of related types of goiter**; those of the myxedematous type with no general symptoms are at one end, and at the other end the forms with general excitement and exophthalmos. Adami believes in the presence of the following conditions of the thyroid, and with symptoms related as indicated: In one case there may be atrophy of the skin, with the usual symptoms of myxedema or cretinism; in the second case the gland tissue may be relatively little affected, but the discharge of the secretion may be interfered with, and the colloid matter is heaped up, with the production of colloid goiter, with associated symptoms of myxedema or cretinism; there may be nodular goiter, with absence of symptoms, except perhaps pressure symptoms; in the fourth case there may be some retention of the material secreted by the gland and occasional attacks of excessive stimulation or altered vascularity of the gland, with consequent excessive absorption or discharge of the material into the circulation. This is the form in which there is ordinary goiter with paroxysmal symptoms of Graves' disease. In the fifth form there may be increased discharge due to retention. This gives a secondary Graves' disease. In the sixth form there may be increased activity of the gland with hyperplasia, which is primary Graves' disease with retention. In the seventh form there may be indistinct signs of Graves' disease without any recognizable changes in the thyroid. These are the incomplete forms of Graves' disease, the "formes frustes" of the French.

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[1] Soc. Rep., Münch. med. Woch., 1899, No. 41, p. 1357.

[2] Beiträge z. klin. Chir., vol. XXIV, No. 3, 1899.

[3] Zeit. f. klin. Med., Bd. XXXVIII, 1899, p. 169.

[4] Glasgow Med. Jour., vol. LII, No. 3.

[5] Montreal Med. Jour., Jan., 1900.

M. Faure [1] reports a case in which there was **combined exophthalmic goiter and myxedema,** the symptoms of the former condition having appeared 33 years before the report. The woman had grown progressively worse until about 1894, when there was decrease in the size of the thyroid and some improvement of the symptoms, but ultimately she exhibited decided swelling of the face, and her memory was poor. She had, however, a tremor, with some prominence of the eyes, and a small hard goiter; her pulse was 100 to the minute and somewhat irregular. Iodothyrin caused quite decided improvement, reducing the rapidity of the pulse and the prominence of the goiter; but afterward she had attacks resembling tetany, and diarrhea with cardiac weakness. She died, and at postmortem the thyroid gland was found to be transformed into a firm adenoma weighing 100 gm. There was apparently some compression of the cervical sympathetic, but there were no evident changes in the nervous system. The heart was hypertrophied.

## MYXEDEMA.

A. Hymanson [2] describes a case which he considered to be a combination of **acromegaly and myxedema.** It occurred in a woman of 39; there were the appearances of the face, chest, and hands commonly seen in acromegaly, but with this there was some hard edema in certain areas, though the skin was in great part soft. Radiographs demonstrated thickening of the soft parts and of the bones as well. Thyroid and pituitary extracts were used without benefit.

E. Ponfick [3] describes 2 interesting cases of myxedema in which advanced **changes in the hypophysis** were found associated with disease of the thyroid gland. The first case ended fatally from pneumonia

[1] Presse méd., Sept. 23, 1899.

[2] Med. Rec., July 1, 1899.

[3] Zeit. f. klin. Med., Bd. XXXVIII, Nos. 1, 2, u. 3.

when the myxedema was not far advanced. There was therefore opportunity to observe the conditions much earlier than is usually the case. It was found that the thyroid was small, the glandular tissues were largely destroyed, and the interstitial tissue was increased. The changes in the hypophysis, however, while of much the same general character, were more marked; the glandular tissue was almost completely destroyed, the connective tissue was largely increased, and the presence of small concrement-like particles in the gland seemed to demonstrate that the process was even older here than in the thyroid. Advanced changes in the hypophysis were found in the second case also, and Ponfick considers that the relation of changes in the hypophysis to myxedema must be held in mind in considering the etiology of myxedema. If any such relation exists, however, it is as yet unproved.

C. W. Chapman [1] reports 3 cases of myxedema which were seen in the early stages, and were extremely difficult to diagnose, the first showing for a long time practically nothing but severe obstinate anemia, mental dullness subsequently appearing and being the first suggestion of the myxedema which afterward developed. Anemia was for a long time the only distinct symptom in the second case also. In the third case the early symptoms were chiefly pain in the precordia and dyspnea. Chapman speaks of the difficulty in diagnosing this condition early, and mentions a **solid appearance of the conjunctiva** as a frequent early sign of value.

D. Sommerville [2] reports a case of **brain tumor** in which, besides headache, loss of memory, weakness of the lower extremities, and a tendency to fall backward, there was a myxedematous appearance of the skin, with extreme deformity of the hands, slow pulse, and a general slowness of perception. As eye changes and vomiting were absent, the diagnosis between myxedema and brain tumor was difficult. The use of thyroid extract caused decided improvement within a few weeks, but the man afterward grew worse, became paralyzed, and died. Postmortem examination showed the presence of a large glioma in the right occipital lobe of the brain, while the thyroid gland was found normal.

R. C. Leeper [3] reports a number of cases treated by **thyroid extract.** The first was one of myxedema in which there were marked mental symptoms. For 2 months thyroid had practically no effect, but subsequent to this there was improvement, which continued, becoming very pronounced. In one other case there was enlarged thyroid, and in a third an apparent reduction in the size of the thyroid. In both the use of thyroid extract caused decided improvement when other medication had failed.

## MISCELLANEOUS METABOLIC AFFECTIONS.

A. Albu [4] discusses the metabolism of albumins in **protracted insufficient nourishment** and the possibility of causing an actual retention of albumins in this condition by giving excessive amounts of proteids. Albu does not believe that it has ever been proved that excessive amounts of proteids, when given to normal persons, cause an increase in the tissue proteids. Krug's experiments, which are the best proof yet offered that such a proteid increase may be thus produced, are faulty in the fact that while experimenting upon himself, Krug was not only taking an excessive amount of proteid, but also increased amounts of fats and carbohydrates, which are known to cause a decrease in the proteid destruction. Albu reports his own experiments in a number of cases in which the nourishment taken had been decidedly insufficient and the muscular tissue had been much reduced. In all these cases he was able to cause a marked nitrogen retention by giving largely increased amounts of proteids. The nitrogen retention had, however, practically disappeared at the end of about 2 weeks, and Albu believes that an equilibrium is established, even in cases insufficiently nourished, after about this time. In his experiments he used a dietetic preparation called plasmon to increase the proteids taken, choosing this because it is a palatable and cheap preparation which is readily absorbed. He believes that, particularly in the poorer classes, the lack of proper amounts of albuminous food is a very common cause of chronic ill health, and that very good results in such cases can be achieved by providing sufficient amounts of readily digested albumins.

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[1] Lancet, Sept. 30, 1899.

[2] Brit. Med. Jour., Jan. 20, 1900.

[3] Ibid., Jan. 27, 1900.

[4] Zeit. f. klin. Med., Bd. XXXVIII, Hefte 1, 2, u. 3.

T. R. Offer,[1] after investigation, concludes that **alcohol does preserve albumin,** though this effect is but slight. He believes, however, that it has a marked protective action upon the fat destruction, and that by this means it may perhaps guard the albumin from destruction.

J. Marischler[2] describes a curious case in a boy of 14 in which there had been since early childhood **profuse sweating** of the upper half of the body over the chest, back, and face. The sweating was more pronounced when the temperature was low. There were no general symptoms except some mental depression. The metabolism was investigated and nothing abnormal was discovered except some reduction of the urinary chlorids, and the lack of chlorids was explained by their elimination in the sweat. The condition seems to be an abnormality of the sweat-centers.

L. Bruns[3] reports an instance of **diffuse scleroderma** of the legs, the upper boundary being sharply circumscribed and corresponding to the nerve distribution of the spinal segment. Because of this curious limitation, and because the disease often shows evidence of trophic changes, Bruns believes that the affection arises in changes in the spinal cord.

Wetzel[4] describes a case of **osteomalacia** in a married woman of 41 who had borne her only child in 1875. The disease was of 10 years' duration, and had been of increasing severity, the patient finally becoming bedridden and scarcely able to move. During a pelvic examination the right femur was broken. The woman ultimately died of peritonitis resulting from fecal retention. Berger[1] describes a case of rapidly progressing osteomalacia in a man 20 years of age. The disease caused great deformity of the extremities and showed no response to treatment of any kind.

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[1] Wien. klin. Woch., Oct. 12, 1899.

[2] Berl. klin. Woch., July 27, 1899.

[3] Deut. med. Woch., July 27, 1899.

[4] Münch. med. Woch., Aug. 8, 1899.

## DISEASES OF THE BLOOD.

### CONDITIONS AFFECTING THE CONSTITUTION OF THE BLOOD.

Reineboth and Kohlhardt[2] report some blood changes which they find to be the result of **exposure to cold.** Rabbits were immersed in water at a temperature of 2° C. for 5 minutes. They found that this caused a decrease in the hemoglobin which persisted for several days; and that if they were subsequently immersed, a decrease occurred again. In one case there was marked enlargement of the spleen. Spectroscopic examination of the blood-serum was carried out, and hemoglobin was determined to be present. They therefore decided that exposure to cold tends to produce hemoglobinuria.

E. Grawitz[3] severely criticizes the results obtained by Reineboth and Kohlhardt. Grawitz's previous work has given contrary results, and he considers the work of the authors mentioned to be untrustworthy largely because they used the Fleischl hemoglobinometer and investigated blood abstracted from constricted veins. Grawitz considers that the results with the Fleischl instrument are not trustworthy, and that constriction of the veins leads to entirely unreliable results. He has repeated his previous experiments by dropping rabbits into iced water and examining the blood taken from unconstricted veins. He found **no evidences of hemoglobinemia** by examination with the unaided eye or with the spectroscope. The serum increased to some extent in specific gravity, and the same phenomenon was observed in the total blood. This Grawitz attributes to exudation of lymph from the capillaries and the blood-vessels into the tissues, and he suggests that the effect of cold baths and subsequent warm douches is largely attributable to exudation of serum and its subsequent return into the blood-vessels, this causing a **flushing of the tissues** and alterations in metabolism.

Reineboth,[4] in answer to Grawitz's criticisms, states that the spectroscopic examination of the blood is not trustworthy. He considers the results obtained with the hemoglobinometer more satisfactory. He also considers that Grawitz in his experiments did not use sufficiently cold water. Grawitz answers that the water used in his experiments was iced water, and he repeats his previous criticisms.

Spangaro,[5] in some investigations concerning the **coagulation of the blood,** learned that if the blood of mammals was taken directly from the vessels it coagulated, though after a longer time than when it had previously been allowed to come in contact with the tissues. Likewise, if the blood were placed in clean vessels, it took longer to coagulate than if small portions of muscle or other tissue were dropped into the vessel or rubbed against the wall of the vessel. Retraction of the clot and separation of the serum occurred with a rapidity corresponding to the rapidity of coagulation. In the blood taken from animals without allowing it to touch the tissues he always observed marked changes in the blood plates and diminution in their number. As the blood is about to coagulate the blood plates collect together, and the same occurs to some extent with the red corpuscles. These changes are much more marked in blood that has touched the tissues. He saw no marked change in the leukocytes after removal of the blood.

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[1] Presse méd., No. 52, 1899.

[2] Deut. Arch. f. klin. Med., Sept. 29, 1899.

[3] Centralbl. f. innere Med., Dec. 18, 1899.

[4] Ibid., Jan. 20, 1900.

[5] Riforma Med., 1899, Nos. 169 and 170.

W. H. Thompson, Jr.,[1] in reporting upon the effects of **injection of peptones** into the circulation, states that amphopeptone delayed coagulation, while coagulation was increased by antipeptone. Both results were occasionally produced by protoalbumose. The vessels were dilated by all the constituents of Witte's peptone except the antipeptone, the dilation being due to paralysis of the peripheral branches of the splanchnics. The effects of Witte's peptones he considers to be due almost entirely to protoalbumose. The most marked effects upon the vessels were seen in those of the abdominal cavity, especially in those of the liver.

Phisalix [2] made some interesting observations concerning the **coagulation of blood in reptiles.** If the blood is allowed to stand, and the clear serum above the corpuscles drawn off, the serum at once coagulates; if, however, a few blood-corpuscles are drawn off with it, it remains fluid. It seems evident, therefore, that the corpuscles contained some substance which interfered with coagulation. If the red blood-corpuscles were subjected to a temperature of 58° C. for 15 minutes, coagulation was hastened.

Hubbard,[3] in reporting the results of his experiments concerning the **coagulating power of blood-serum,** stated that he had investigated various serums in living persons, chiefly using the antistreptococcic, antidiphtheric, and antisyphilitic serums. Their coagulating effects are independent of the antitoxic properties. In one case of aneurysm of the aorta the antisyphilitic serum caused a reduction of the pain, which had been severe, and improvement in the other signs. When the pain returned a month later, further injections of serum caused renewed improvement. He had also used antidiphtheric serum with success in menorrhagia, and in a case of hemophilia with severe epistaxis antistreptococcic serum and antidiphtheric serum were both used, and the hemorrhage was completely and permanently controlled. Normal serum had been used also, but had given no definite results. He believes that the antidiphtheric serum probably has more active coagulating powers than the others.

A. Christomonos [4] **produced hemoglobinuria** by the injection of glycerin into rabbits, and then investigated the **fate of the red corpuscles.** The effect of the injection was an apparent increase of the red cells, followed by an actual diminution in their number, the absorption of water by the glycerin producing the primary apparent increase. Soon after the injections hemoglobin was abstracted from the red cells; the corpuscles, however, often circulated for some time after losing much of their hemoglobin. Finally they were apparently destroyed by the spleen and bone-marrow, but the last changes were unknown. The kidneys showed a large amount of blood pigment in the tubules when there was hemoglobinuria, and at the same time a great deal of water was withdrawn from the blood; so large an amount was sometimes abstracted that finally the kidney tubules seemed to be almost filled with hemoglobin, and the hemoglobin occasionally formed tube-casts, while the excretion of urine became much reduced and there was practically anuria. The kidneys swelled and the animals had pain and edema. There were no changes found in the liver.

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[1] Jour. Physiol., vol. XXIV, No. 5; vol. XXV, No. 1.

[2] Compt. rend. de la Soc. de Biol., Nov. 11, 1899.

[3] Lancet, Oct. 4, 1899, Society Report.

[4] Virchow's Archiv, Bd. CLXI, S. 582.

A. Biowicz,[1] after the injection of hemoglobin solution under the skin of dogs, observed after 5 hours characteristic **crystals of hemoglobin in the liver-cells.** He also believed that he observed erythrocytes and other products within the liver-cells.

G. M. Malkoff,[2] in discussing the question of **agglutination of the red blood-corpuscles,** states that he proved by repeated investigations that the blood of guinea-pigs and doves has no effect upon the blood-corpuscles of goats; but if the guinea-pigs or doves are first injected with defibrinated goat's blood, their serums show marked action on the red corpuscles of the goat. This is evidently a specific action. It occurs practically only with the blood of the goat, and not, for instance, with human blood; and that it is due to some specific substance which unites with the red corpuscles is shown by the fact that Malkoff found that if he added to goat serum the blood of doves, rabbits, and man, all would rapidly agglutinate. If one of these forms of blood were first added, and then the mixture were centrifugated, the remaining serum acted only upon the bloods that had not been previously mixed with it, and not upon that with which it had already reacted. If all three bloods were added, it would not afterward react to any of them, showing that there are certain agglutins present in the blood corresponding in number and kind to the number of forms of cells with which the blood will react, and that the agglutins are specific bodies which have affinity only with the peculiar morphologic element which they cause to agglutinate.

Rosin and Jellinek,[3] in discussing the **color strength and iron** of human blood, report the investigation of the relation of these two points to the hemoglobin. They investigated a series of cases, and note a number of facts as a result. In valvular cardiac lesions, particularly when there was loss of compensation, there was an exceedingly high color with decrease of iron. The color was high in jaundice, diabetes, and Graves' disease, while the iron was low. The color was lessened in chlorosis and other forms of anemia, but the iron was relatively high. In leukemia the iron was diminished and the color about normal.

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[1] Bull. internat. de l'Acad. des Sc. de Cracovie, July, 1899.

[2] Deut. med. Woch., April 5, 1900.

[3] Zeit. f. klin. Med., Bd. XXXIX, p. 109.

A. Jolles [1] has investigated the question of the **presence of iron in the blood-serum** in a series of cases, using his ferrometer as a method of determining the presence of iron. Eighteen normal persons gave negative results, as did 3 of chlorosis, 1 of leukemia, 1 of interstitial nephritis, and 1 of neoplasm. In 2 cases of severe anemia, however, he found traces of iron in the serum, and there were determinable quantities in 2 cases of severe diabetes. From the latter result Jolles thinks it may be possible to prove that some importance may be attached to the presence of iron in the blood-serum in **diabetes.** [The method, however, seems to be very unsatisfactory, since in carrying it out the blood is diluted with 0.8 % salt solution and then centrifugated. There is an evident error in this, as the diluting solution remains of constant strength, while the concentration of the blood varies in different cases. The difference in the molecular concentration of the diluting solution and of the blood used might readily result in destruction of the blood-corpuscles and consequent presence of iron in the serum.]

Gottstein,[2] in discussing the change in the number of blood-corpuscles at **high altitudes,** states that he has abandoned his view that the changes in the counts are due to alterations in pressure on the cover-glass of the counting apparatus, but he still believes that the change in count is apparent and not real, and is due to some mechanical difficulty with the apparatus. To prove this he made a suspension of yeast-cells in formalin solution, and determined the number of cells by the hemocytometer in repeated counts. He then went to different altitudes and saw increase in the count as the altitude increased. The apparatus must have been at fault, but in what way it is difficult to say.

F. Aporti,[3] from his clinical observations in Riva's Clinic, has come to believe that the use of **hemoglobin** in anemia causes an **increase** in the amount of **hemoglobin** only, the red cells not being affected in number, while **arsenic** causes an **increase in the red cells** without any increase in the hemoglobin. His experimental work also led to the same conclusion. This work he carried out by repeatedly abstracting blood from animals and keeping them on iron-free food. As a result of the blood abstraction he found that there was constant effort on the part of the system to produce new blood-cells, and for a long time new hemoglobin was formed, the iron, of course, coming from the iron deposits in the organs, particularly from the liver. If, however, the abstractions of blood were carried out for a considerable period, the iron of the organs finally became exhausted, new hemoglobin was no longer formed, and the animal died. When he estimated the amount of iron in the various organs of such animals, he found the amount in the liver greatly reduced as compared with normal animals, the quantity being about the same as that in the other organs. The whole amount of iron in the organism

[1] Centralbl. f. innere Med., July 1, 1899.

[2] Münch. med. Woch., Oct. 3, 1899.

[3] Centralbl. f. innere Med., Jan. 13, 1900.

was very small. When, however, the abstractions of blood were carried out for a time and only iron-free food was given, and when at about the period that the animals had been brought to the point of exhaustion of the tissue-iron they were given arsenic, the red cells increased, but the hemoglobin did not; if iron were then given, the hemoglobin increased with very great rapidity. In one case the hemoglobin increased from 50% to 95% in 7 days. Aporti considers that the production of hemoglobin and of the red cells depends upon wholly different factors, some substances increasing one, while others increase the other.

K. Gregor[1] made some experiments concerning the **amount of ammonia in the blood** in experimental acid intoxication. Animals were given, 3 to 6 times a day, from 200 cc. to 250 cc. of a solution containing 8 parts of hydrochloric acid to 1000 of water. The amount of ammonia in the peripheral blood was then investigated, and control estimations were made in untreated animals. There was no evidence that the acid produced any increase in the ammonia of the blood.

D. H. Jones[2] reports 53 cases tested by **Justus' blood test for syphilis**; 18 were control cases, and all of these were negative. Of the 35 syphilitic cases, 17 were active syphilis not under treatment, and 13 of these gave positive results. One active case under treatment gave a negative result. Of 8 cases of chancre with adenitis, 2 reacted positively and 6 negatively. Both the positive cases reacted only to repeated tests. Seven cases of chancre without adenitis gave only 1 positive reaction.

J. Monod,[3] in discussing the anemia of syphilis, states that the changes in the blood provide one with the **first evidences of** the existence of **syphilis.** The early anemia is sometimes of simple form, and sometimes chiefly affects the hemoglobin. The degree of anemia is often in direct correspondence to the appearance and severity of the specific symptoms. Leukocytosis appears early and persists for a long time. It chiefly affects the mononuclear leukocytes. In children the anemia may be associated with the appearance of nucleated red cells and a marked increase of the large mononuclear cells from the bone-marrow, together with some increase of the other forms of leukocytes. The use of mercurial treatment causes a reduction in the number of leukocytes and an increase in red blood-corpuscles and hemoglobin, unless the treatment is excessively prolonged, when the anemia is usually increased in degree. This indicates the necessity for an intermittent use of specific treatment.

**The Red Corpuscles.**—Grawitz[4] describes the presence of peculiar **basophile granules** in the interiors of many of the red cells in a case of tertian intermittent malarial fever. They were not seen after using the triacid mixture, and were satisfactorily brought out with eosin methylene-blue only when the staining was carried out for a long time. Grawitz then investigated a series of cases of varied nature, and found the granules in 2 instances of cancer, 2 of leukemia, and in a number of cases of pernicious anemia and of sepsis. In examining 5 healthy persons, they were found absent in every instance; likewise in 2 cases of tuberculosis, 1 of chlorosis, 1 of diabetes mellitus, 1 of measles, and 1 of anemia pseudoleukæmica. Grawitz thinks that these granules are not identical with those which have been previously described by Plehn or those reported by Heintz and Ehrlich. They are also not polychromatophilic degeneration, since they always take a pure blue stain. Since no changes resembling these were found in the cells in the bone-marrow, and there was no other evidence that the cells were the result of degenerative processes, Grawitz concludes that they were the result of degeneration of the hemoglobin. He considers it possible that their presence may prove important in the future in indicating that blood changes in individual cases in which these granules are seen are due to a destructive agency rather than to abnormal degenerative changes.

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[1] Centralbl. f. Allg. Path. und pathol. Anat., 1900, No. 1.

[2] N. Y. Med. Jour., April 7, 1900.

[3] Thèse de Paris, 1899, 1900.

[4] Deut. med. Woch., Sept. 7, 1899.

M. Kohn [1] discusses the basophile granulations of the blood-corpuscles. They were absent in splenic anemia. The granules appeared after abstracting large quantities of blood from animals, but their appearance was delayed about 24 hours. Kohn thinks that this indicates that they are **produced by degeneration** of the protoplasm of the cells, probably chiefly as a result of hydremia.

A. Grawitz,[2] in discussing the basophilic granulations of the red corpuscles, states positively that he considers them evidences of degeneration, as they are not found preformed in bone-marrow, and the granulations evidently do not result from karolytic changes in the nuclei. He found them in considerable numbers in **lead-poisoning.** They were absent in chlorosis, tuberculosis, and syphilis. They were absent in Bright's disease and in cirrhosis of the liver unless there was severe cachexia. Their presence in large numbers indicates an intense anemia. Grawitz considers the granules discovered by Plehn in Europeans dwelling in the tropics the result of degeneration caused by intense heat, since he has been able to produce similar changes in the blood of mice by subjecting them to high temperatures.

G. Schmanch [3] noted the presence of **bodies within the red blood-corpuscles** of the cat. These were seen in the blood of 18 cats, both in the fresh blood and after adding methyl-violet. They were quite numerous. The size and shape varied. They showed movement in normal salt solution, but Schmanch does not think that they are parasites; he considers them remnants of the nuclei. The nuclei of normoblasts may be observed to show chemical changes when these cells enter the blood current, oxychromatin replacing the basic chromatin, and ultimately the chromatin being absorbed by the hemoglobin-containing portion of the cell and undergoing solution. The parasite-like bodies observed could be increased by the use of pyridin or the extract of bothriocephalus, both of which substances produce anemia and contain toxic substances.

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[1] Münch. med. Woch., Feb. 6, 1900.
[2] Berl. klin. Woch., Feb. 26, 1900.
[3] Virchow's Archiv, Bd. CVI, S. 201.

**The Leukocytes.**—J. Arnold[1] investigated the question whether the **granular bodies found in the cytoplasm** of the corpuscles are merely granular excretion products or are preformed structures and a living part of the plasmosome. His method was to introduce thin plates of elder pith into the lymph-sacs in the backs of frogs, leaving the pith in position for from 6 to 48 hours. Hematogenous wandering cells were found in the pith when it was removed, and various stains were used on these corpuscles, the most satisfactory being neutral red and methylene-blue. Arnold found that the granules were readily seen before staining, and that they were present in the cells which showed changes of form and active movement, the cells being, therefore, evidently living. The color which they took upon staining was sometimes that of the methylene-blue and sometimes red. The granules were evidently not merely particles taken up by phagocytic action, nor were they the result of precipitation during staining. The fact that some of them were stained and some were not, and that intermediate pictures between the stained and unstained particles were seen, that some were large and some small, and other factors, leads Arnold to believe that they are active parts of the plasmosome, some of them having undergone change. Results similar to those obtained in frogs were found in experiments on rabbits. Arnold believes that the granular structural elements of the cells should be called **plasmosomes,** while the granular products of metabolism alone should be termed **granules.** Probably the latter are produced from the former.

O. Naegeli[2] notes that there are **3 forms of cells free from granules** which are found in the bone-marrow: a small form resembling lymphocytes; a somewhat larger form with slightly paler nucleus; and, finally, cells which, except for the absence of granules, are exactly like myelocytes. Naegeli believes that it is an artificial classification to divide these 3 forms into classes, because intermediate stages between all of them may often be seen. He believes that they are not related to lymphocytes at all, and calls them **myeloblasts** because myelocytes are formed from them. As to the difference between them and lymphocytes, he mentions a tendency to a marked difference in the size and the frequency of oval forms, while lymphocytes are always round; the nucleus in these cells is often oval, and chromatin is present in marked degree and is always arranged in a regular net-like form, while in lymphocytes it is irregular. The myeloblasts do not contain nucleoli, while lymphocytes do. The protoplasm of lymphocytes is much more distinctly basophile. Another important point is the fact that the blue color given by tincture of guaiac in bone-marrow is not given by lymph-glands and other organs containing large amounts of lymphocytes, and apparently the guaiac reaction occurs most decidedly in those cases in which there are large numbers of myeloblasts. He believes the conditions in pernicious anemia offer further evidence that these cells are distinct from lymphocytes, for the white cells of the bone-marrow in pernicious anemia are almost exclusively of this form and it is recognized that the bone-marrow reverts to embryonic condition in pernicious anemia.

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[1] Virchow's Archiv, Bd. CLVII, p. 124.

[2] Deut. med. Woch., May 3, 1900.

P. Lengemann [1] describes his results in some work on the effect of **injections of particles of tissues,** chiefly in their relation to the **origin of leukocytosis,** and of cell emboli from the bone-marrow. He has repeatedly observed the nuclei of giant cells in the lungs, yet has never seen giant cells with protoplasm, and he believes that the nuclei, and not the cells, are carried to the lungs, and that they do not reach the lungs by their own motion, but by being washed out from the bone-marrow. He bases his belief upon the fact that the cells themselves are not found in the lungs, that nucleated red blood-corpuscles were frequently found in the blood current of the animals experimented upon, and particles of bone-marrow tissue were at times found in the lungs. Also the changes in the bone-marrow indicated that the cells were washed out; after the injections the marrow became much softer and more fluid than normal, and there was an increase in the size and number of blood spaces, with a diminished number of leukocytes. The leukocytes also frequently projected into the spaces or were found free in them, as if they were being washed away. He thinks that normally there is a communication between the arterial and venous capillaries through the spaces occupied by the collections of leukocytes, but if the bone-marrow becomes very hyperemic, the flow through the spaces becomes so forcible as to wash away leukocytes, nucleated red cells, and giant cells. The ingestion of leukocytes by giant cells which is sometimes seen he considers to be accomplished by the fluid protoplasm of the giant cells flowing out about the leukocytes, surrounding them, and then contracting, thus inclosing the leukocytes within the giant cell protoplasm.

K. Brandenburg [2] found that **tincture of guaiac** turned blue with a chloroform extract of **pus.** It has been known before that pus gives a blue reaction with guaiac. The substance in the chloroform extract which caused the reaction was found to be a nucleoproteid, which was probably contained in the leukocytes. The liver, spleen, thymus, and lymph-glands gave no reaction, but the reaction was produced markedly by bone-marrow. The nucleoproteids of the urine gave no reaction, hence they seem not to be the product of leukocytes, but of tissue cells. The guaiac reaction may be used for the demonstration of pus in the urine, but certain other substances may be present in the urine which will interfere somewhat with the reaction, and it is best to carry it out by filtering the urine and applying the guaiac to the filter-paper.

E. G. Horder,[3] after studying the so-called **blood dust,** decides that it is **merely granules** which have been extruded from the leukocytes, and believes that instead of blood dust, these substances should be called blood granules.

L. Grünwald [4] discusses the so-called **hypeosinophile granules.** These granules are peculiar in that they stain with eosin, are decolorized by acids and alkalies, and take a deep fuchsin-red with the triacid mixture. They are found in the round cells of the sputum, pus, seropurulent exudates, and inflammatory tumors; he also observed them in the blood, but only when the preparations were dried in air. They were seen best when the blood preparations were fixed with 1 % formol-alcohol, and were not well seen when the fixing was done with alcohol and ether, bichlorid, or heat. When these methods of fixing were used, the granules commonly had the appearance of ordinary eosinophile granules. They were present in both the polymorphonuclear and mononuclear leukocytes. The best stain is alcoholic solution of eosin with a counterstain of methylene-blue. The staining must not be protracted or the granules will be decolorized.

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[1] Deut. med. Woch., Dec. 28, 1899.

[2] Münch. med. Woch., Feb. 6, 1900.

[3] Lancet, Oct. 14, 1899.

[4] Centralbl. f. innere Med., July 29, 1899.

Bettmann [1] believes that the granules described as hypeosinophile granules are purely the **result of the method of fixation.** He has noted, he states, that when preparations from the same blood are allowed to dry in air, these granules appear; while when fixed by Ehrlich's method, they appear as neutrophile granules. He also notes that neutrophile granules disappear in stagnating pus, and he believes that the changes in the granules are the evidences of degeneration. He thinks that it is exceedingly important in reaching conclusions from the observation of morphologic alterations in the blood-corpuscles that the same technic should always be used. He admits that Grünwald's technic may be quite as good as Ehrlich's, but thinks that positive conclusions can not be drawn from the results of either method, as but little is yet known as to the actual nature of the different granules and the causes of alterations in them. Grünwald,[2] in reply to Bettmann's statement, denies that it is possible to make the same granules take at times a hypeosinophile stain and at other times a neutrophile stain. [Our own experience has been that the granules under discussion sometimes assume neutrophilic and at other times eosinophilic properties, according to the mode of preparation.]

L. Hofbauer [3] investigated the occurrence of **iodophile granules** in the leukocytes in a series of diseases. The method which he recommends is placing the smear preparation blood-side downward upon a solution consisting of 1 part iodin, 3 parts potassium iodid, 100 parts distilled water, and sufficient gum-arabic to give the mixture a syrupy consistency. After one minute the excess of the stain which adheres to the preparation is removed as completely as possible with filter-paper, thus avoiding overstaining. He found the granules present in cases of grave anemia of bad prognosis, while in milder forms they were absent; thus they were not seen in 17 cases of chlorosis or in 18 cases of mild secondary anemia, but were almost constantly seen in 7 cases of grave secondary anemia and in 5 of pernicious anemia. He decides, therefore, that their presence is of evil prognostic import. The granules were seen in all of 9 cases of leukemia, but were absent in pseudoleukemia and in anæmia infantum. He also notes that in a case of hemorrhagic purpura large numbers of extracellular iodophile particles were seen, and he considers that this observation supports the statement of Goldberger and Weiss that the extracellular iodophile particles are increased in cases in which there are hemorrhages into the tissues. He found these granules in numerous cases of pernicious anemia and grave secondary anemia in which there was no leukocytosis, contrary to the statement of Liveriato that they are seen only in cases which show leukocytosis.

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[1] Centralbl. f. innere Med., Feb. 3, 1900.

[2] Ibid., April 7, 1900.

[3] Ibid., Feb. 10, 1900.

Widal and Laisne [1] describe a case of general enlargement of the glands of the mediastinum, neck, and axilla, in which examination of the glandular masses showed large numbers of eosinophiles, and the blood during life presented **26% of eosinophiles.** Shortly before death eosinophilia diminished, and finally the eosinophiles disappeared completely from the blood. In discussion Achard stated that eosinophilia is common in persons who have general glandular enlargement. He cited a case of lymphosarcoma in which there was marked eosinophilia of the glands without eosinophilia of the blood.

Fuchs,[2] in investigating the **sputum** for **eosinophile cells,** found them in all of 25 cases, which included active and passive catarrh of the bronchi, bronchiectasis, and infarct of the lung. He has always seen them in pleural and peritoneal effusions. They were present in large numbers in a case of pulmonary tuberculosis shortly before death. The result of his study of cases and of the literature was to lead him to conclude that these cells arise in various ways, some of them coming from the neutrophiles, some of them being produced by phagocytosis, some of them from altered erythrocytes; they may arise in any situation. He considers that those which are found in the sputum are probably found in the respiratory tract. They have no special diagnostic import except that they appear in diseases that are not associated with fever, occurring only in convalescence from febrile disease.

## METHODS OF EXAMINATION OF THE BLOOD.

D. D. Scannell [3] describes the use of **Oliver's hemocytometer,** and gives the results of control estimations with this and with the Thoma-Zeiss instrument. With the exception of 2 cases in which there was marked leukocytosis, the results differed only as much as 74,000 in the count, the least difference being 4000 and the average difference only about 35,000. This is within the error of the Thoma-Zeiss instrument. It had been known previously that a marked leukocytosis causes an error in the reading. This Scannell also observed.

B. M. Linnell,[4] in discussing the various **methods of estimation of hemoglobin,** expresses dissatisfaction with color tests. He finds Hammerschlag's method much more satisfactory. In order to determine the variations in this method which may take place at various hours during the day, he made estimations of the specific gravity during 16 hours in a healthy subject, 21 years of age, before and after meals and at other periods. He found but slight variations, the usual specific gravity being about 1061; variations between 1058 and 1063 being found.

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[1] Gaz. des Hôp., July 7, 1899.

[2] Deut. Arch. f. klin. Med., Aug. 18, 1899.

[3] Boston M. and S. Jour., Feb. 15, 1900.

[4] Jour. Am. Med. Assoc., July 1, 1899.

A. Jolles [1] describes a simplified form of his **ferrometer.**

Drazo [2] has found the sodium chlorid method of determining the **isotonicity** of the blood to be unsatisfactory. In investigating a series of other substances as to their importance in this connection, he reached the conclusion that **solutions of iodin** act best. He used 2 stock solutions of very different strengths, and mixed them with distilled water, thus obtaining a final solution identical with the specimen investigated. The solutions recommended are: first, one containing 18.75 cg. of metallic iodin, and 17.5 cg. of potassium iodid, dissolved in 100 cc. of distilled water; second, one consisting of 1 cg. of metallic iodin, 2 cg. of potassium iodid, and 40 cc. of distilled water.

E. J. Horder [3] prepares blood smears by touching a drop of blood as it exudes from the puncture with a small square piece of gutta-percha held in a forceps, and immediately smearing this over a cover-glass held in another pair of forceps. He states that by this method work can be done very readily and with very satisfactory results. [By the ordinary method, if properly carried out, artefacts can readily be avoided, and this method seems unnecessary.]

L. Michäelis [4] contributes a description of a **universal method of staining,** which makes 2 preparations unnecessary. He places the smears for from ½ hour to 24 hours in alcohol or fixes on a copper plate. The stains then used are made up from 2 stock solutions, the first of which consists of a 1% solution of chemically pure methylene-blue; the second is a 1% solution of chemically pure eosin. It is important that the distilled water used in making the solution should be fresh and pure. This is determined by adding a drop of hematoxylin to a portion of the water; the result should be a yellowish tinge; if a purple color is seen, the water should be discarded. In staining one takes 20 cc. of absolute alcohol and 20 cc. of the first solution, also 20 cc. of acetone and 12 cc. of the second solution; then 1 cc. of each of these new solutions is taken and the two small portions are mixed immediately before the staining is done. The smear should then be placed upon this mixture with the blood-side downward and allowed to sink to the bottom and to remain there for from ½ minute to 10 minutes. The point of chief importance in the staining is that the first color assumed is blue, and this is afterward replaced by red; the staining should be stopped at once when the red color has completely replaced the blue. If the preparation is too blue, another should be stained for a longer time; but if it is too red, and the blue has vanished, another preparation should be stained for a briefer period.

H. Hewes [5] carries out a **general staining of the leukocytes** by the use of Ehrlich's triacid mixture for 4 minutes, washing in water, and then staining for a few minutes with Loeffler's alkaline methylene-blue. He states that in this manner one may stain malarial organisms, and also procure a good stain of both the granules and the nuclei of the leukocytes and a clear distinction between nucleated red blood-corpuscles and lymphocytes. [We have used the same method with satisfaction for some time.]

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[1] Berl. klin. Woch., Oct. 30, 1899.

[2] Riforma Med., Nos. 173, 174, and 175, 1899.

[3] Lancet, Sept. 30, 1899.

[4] Deut. med. Woch., July 27, 1899.

[5] Boston M. and S. Jour., July 13, 1899.

A. E. Taylor[1] used hematoxylin-eosin and then Loeffler's methylene-blue for a very short time in order to secure a good general stain.

A. Plehn,[2] in **staining** for the **karyochromatophile bodies** in the blood of persons living in malarial districts, uses Ehrlich's hematoxylin-eosin mixture for from 8 to 12 hours, preparations being placed in a covered dish in order to avoid the loss of acetic acid from the stain. The preparations are then washed, dried, and immediately mounted in balsam.

## CHLOROSIS.

F. Forcheimer,[3] in a paper on the **toxicity of the urine,** retracts the statements which he previously made concerning a toxic body in the urine in chlorosis. He now considers the discovery of this toxic substance the result of faulty methods, and does not believe that it can be considered to have any value in relation to the origin of chlorosis.

Leichtenstern[4] contributes an interesting paper on **venous thrombosis in chlorosis.** In 1653 cases which he has seen in the past 15 years thrombosis was seen 11 times, and probably a considerable number of other cases had mild thrombosis which could not be positively diagnosed. The occurrence of thrombosis in chlorosis is favored by hemorrhage, obesity, or pregnancy. In a study of the literature he found that in 86 cases the veins of the lower extremities were affected 48 times, the cerebral sinuses 29 times; the latter cases were usually fatal. The small veins of the muscles were not uncommonly affected in the lower extremities; this produces only slight signs, and makes the diagnosis difficult at times, so that serious mistakes may be the result of lack of a proper diagnosis. He states that in 52 cases of thrombosis of the lower extremities pulmonary embolism was observed 10 times, demonstrating that the clots in chlorotic thrombosis are very readily broken up and emboli are likely to form. This is probably due to the fact that the thrombi are rich in blood. The frequency of thrombosis in chlorosis is probably a result of cardiac debility combined with blood changes and alterations in the interiors of the blood-vessels.

**Treatment.**—W. Wolf[5] notes that, following the belief now prevalent that iron acts in chlorosis by exciting the activity of the blood-forming organs, some authors, particularly Carvello and Barabine, have believed that other heavy metals might cause analogous stimulation of the blood-forming organs; the authors mentioned have reported as the result of their experimental work that some of these metals, particularly copper, zinc, manganese, and mercury, do have this effect in chickens. Wolf carried out similar experiments in rats, giving them finely powdered **copper sulphate and zinc,** and continuing the administration of the metals for from 40 to 50 days. Control animals were kept under observation at the same time. Wolf could not confirm the results of the authors mentioned. In comparing the control animals with those that received the metals he found no difference in the body-weight, in the number of corpuscles, or in the amount of hemoglobin.

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[1] "Studies in Leukemia," Contributions from the Wm. Pepper Laboratory of Clin. Med., 1900.

[2] Deut. med. Woch., Dec. 21, 1899.

[3] Am. Jour. Med. Sci., Sept., 1899.

[4] Münch. med. Woch., Nov. 28, 1899.

[5] Zeit. f. physiol. Chem., Bd. XXVI, p. 442.

A. Hoffmann[1] investigated a series of 98 rabbits in regard to the **absorption of iron** and its fate after reaching the tissues. He decided as a result of his work that the iron is practically all absorbed in the duodenum, from which place it is carried away by transport cells and combined with the albumin of the blood. It has no toxic action in this form. From the heart it is deposited in the liver and spleen, and especially in the bone-marrow. He believes that in the bone-marrow particularly it stimulates the physiologic functions and increases the rapidity of transformation of the embryonic red cells into nonnucleated erythrocytes. He believes that preparations of hemoglobin are not more valuable than the older preparations of iron, nor are the various other forms of inorganic iron especially useful, the effect of iron, in his opinion, depending purely upon the amount of the metal absorbed.

W. Nathan,[2] after experimenting on animals with the use of **iron-free food and of iron somatose,** reached the conclusion that the iron somatose was freely absorbed by the small intestine, most of the iron being transported to the central canal and thence to the lymph-system. It is questionable whether any was absorbed by the blood-vessels. Most of the iron was excreted in the large intestine by means of the leukocytes, but some was present in the central canals, and it is possible that some absorption took place in the large intestine also. There was no evidence of excretion of iron from the kidneys.

Steffanelli,[3] after an investigation of the value of the **hypodermic use of iron, manganese, and arsenic** in the treatment of chlorosis, decides that iron causes menstruation to be established fairly early as a result of the general improvement caused by the drug. Manganese seems to have a more marked effect upon the general health, but does not influence the menses so quickly. Iron and manganese, he believes, act chiefly upon the hemoglobin, but also upon the red cells. Arsenic has no definite effect upon the hemoglobin, but does increase the number of red cells.

Grocco,[4] in discussing the use of iron by **hypodermic and intravenous injections,** states that the latter form of administration is no more useful than the hypodermic, and is dangerous. Hypodermic injections are well borne, and are usually efficient. They are very valuable when iron is not well borne by the stomach or does not produce good effects. Soluble preparations from manganese may be given hypodermically in place of iron.

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[1] Münch. med. Woch., 1899, No. 29.

[2] Deut. med. Woch., Feb. 22, 1900.

[3] Settimana Med., Nos. 40 and 41, 1899.

[4] Ibid., Feb. 3, 1900.

O. Jollasse [1] has found that the administration of the citrate of iron in **starch enemas** resulted in good absorption, and had apparently about the same effect as iron taken by the mouth. This method of administration caused some abdominal pain, but no other unfavorable results, and he considers that it may be used with satisfaction when the stomach rebels against the administration of iron.

## PERNICIOUS ANEMIA.

**Etiology and Pathology.**—J. G. Adami [2] has found in the livers of patients who had died of pernicious anemia **organisms** similar to those which he has previously described in cirrhosis of the liver. He attributes pernicious anemia to what he calls **subinfection.** This he explains as follows: He considers that normal leukocytes carry bacteria from the digestive tract into the lymphatics and small veins, and especially into the small veins of the portal system. Commonly the bacteria degenerate or are digested, or at times may reach the general circulation, and are excreted by the kidneys; the condition then corresponds practically to that called latent infection by the French, no disease resulting in such instances. Sometimes, however, a larger number of bacteria enter the blood, and there is produced an excessive activity of the glandular system and other organs in the attempt to destroy the microorganisms, and thus chronic inflammatory changes are set up. This condition Adami terms subinfection. To it he has previously attributed cirrhosis of the liver, and he has now come to consider pernicious anemia to be the result of such a subinfection through the gastro-intestinal tract. [These observations are interesting and will merit further study, though they can not as yet be accepted as proved.]

W. Hunter [3] describes 7 cases of pernicious anemia, chiefly in their relation to the view which he now expresses, that the disease is due to **infection from carious teeth.** Hunter has long been of the opinion, which is shared by many, that the disease is an infection, and from the observation of a series of cases he has concluded that the infection begins in necrotic conditions of the teeth. This produces an infective catarrh of the gastro-intestinal tract, leading to chronic alterations in the mucous membranes and to a toxemia, the general symptoms and the hemolysis being the result of the toxemia. He believes that the treatment of the disease should consist largely in careful attention to the condition of the mouth, and particularly of the teeth, using antiseptic washes and gastro-intestinal antiseptics. The use of antitoxic serum will probably be of value.

Lipowski [4] notes the changes that have taken place in **methods of diagnosing pernicious anemia,** fatal progressive anemia being considered the chief point in diagnosing the disease at present. If macroblasts are present in the blood, they are commonly considered to denote the presence of this disease. They are, however, not infrequently absent. The diagnosis of pernicious anemia by the presence of microcytes and poikilocytes is now recognized as impossible. Lipowski puts the question as to whether we can make a diagnosis of pernicious anemia whenever there is present a fatal anemia with the evidences of incurable lesions of the bone-marrow. This question is based upon the observation of the case of a woman of 31 who was admitted to the hospital with profound anemia and hemorrhages from the mucous membranes following the ingestion of an unknown poison. The anemia progressed to a fatal issue. The bone-marrow was believed to be so severely affected that it practically did not functionate, since nucleated red cells were entirely absent and 90% of the white cells were lymphocytes; the latter testimony is considered important because the bone-marrow produces the neutrophile cells, and these were almost wholly absent. Lipowski also directs attention to the fact that the general signs of pernicious anemia may be present and the blood count may indicate the disease before there are any morphologic alterations in the blood which are characteristic of this disease. He describes a case in which there was a progressively increasing anemia, the hemoglobin finally reaching 10%, and the red blood-cells numbering 800,000 at the time of death; there had been hemorrhages in the skin, changes in the eye-grounds, and irregular fever, and a diagnosis of pernicious anemia was made moderately early in the case, but only late in the course of the disease did he observe alterations in the size of the corpuscles, and orthochromatic and polychromatic normoblasts and macroblasts were never seen.

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[1] Münch. med. Woch., 1899, No. 37.

[2] Med. News, Jan. 6, 1900; and Jour. Am. Med. Assoc., Dec. 16 and 23, 1899.

[3] Lancet, Jan. 27, Feb. 3 and 10, 1900.

[4] Deut. med. Woch., May 24, 1900.

**Symptomatology.**—A. Abrams [1] reports 2 cases of pernicious anemia, in one of which the blood count was reduced to 1,000,000 red cells and a hemoglobin of 40%; in the other the red cells numbered 800,000 and the hemoglobin was 35%. In both cases the **gastric symptoms** were so marked as to make it difficult to exclude gastric carcinoma. When given arsenic, however, they both improved rapidly, and apparently, from the report, considerably, one man returning to his work. [The remarkable ability of cases of pernicious anemia to continue at work despite marked anemia is well known, and must not be overlooked.]

J. M. Da Costa [2] reports the case of a man of 47 who had severe anemia, the red cells being reduced to 2,600,000 and the hemoglobin to 17%, while the leukocyte count was 12,000. The red cells varied in size and shape, and nucleated red cells were absent. Because of the latter fact, and of the disproportionate reduction of the hemoglobin and decrease in the white cells, Da Costa considers the case one of grave secondary anemia. The man had had prolonged digestive disturbance even preceding his anemia, and at the time of the report he had achylia gastrica; hence the cause of the anemia was thought to be the protracted **gastro-intestinal disturbance.**

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[1] Med. Rec., April 28, 1900.

[2] Phila. Med. Jour., Dec. 30, 1899.

Schwabach[1] reports upon the **conditions of the ear** as observed in 7 cases of pernicious anemia. The symptoms usually observed were noises in the head and reduction of the acuity of hearing. In only one was there any change in the drum membrane. In this case a small ecchymosis was seen, and this was followed by suppuration and perforation, and later by healing. One case was examined microscopically, and showed extravasation of blood in the middle ear, but absence of changes in the inner ear. The changes were similar to those seen in leukemia, and Schwabach considers that the causes of the changes in hearing are merely alterations in the sound-conducting apparatus, not in the true auditory apparatus.

**Treatment.**—W. Elder,[2] acting upon Hunter's suggestion that pernicious anemia may be due to chronic infection through the alimentary tract resulting from bad teeth, has treated a case of pernicious anemia by using antiseptic mouth-washes and giving **antistreptococcic serum.** When admitted to the hospital, the man's blood count was 797,500 reds, 24% of hemoglobin, 4520 leukocytes. A week later, when the treatment was started, his red blood-corpuscles were 1,962,500 and the hemoglobin was 44%. The serum treatment was continued for 45 days, 18 injections of 10 cc. each being given. At the end of this period a remarkable improvement is said to have occurred. The man was almost well; his red corpuscles were 4,800,000 and his hemoglobin was 104%. The clinical picture of the case is not clearly one of pernicious anemia. The condition of the blood speaks strongly in favor of this, but the man had a great deal of fever, which rose as high as 103.6° F., and had maniacal delirium throughout the whole course of the treatment; even at the end of the report he did not seem quite in his right mind. He had marked diarrhea and there was some blood in his stools, though this had not been present in large quantity. He had had some pain in the region of the liver. The liver was thought not to have been enlarged. His skin was a pale yellow color, which does not seem to have had any resemblance to jaundice, from the report. The course of the case had been only about 6 months, however, and there was so much fever and constitutional and mental disturbance that there is a strong suggestion of an acute sepsis in spite of the blood conditions. Elder notes that this man's teeth were in a very bad condition, and considers this some support for Hunter's view of the etiology of pernicious anemia.

Fowler[3] investigated the value of the use of **red bone-marrow** in anemia by producing anemia in young rabbits through feeding them on diets deficient in iron, in proteids, and in both proteids and iron. The lack of iron caused a pronounced reduction in hemoglobin, while lack of proteid caused the red cells to decrease largely; pure carbohydrate diet caused the most severe alterations in the blood. After animals had been made anemic they were given bone-marrow. This substance had no effect upon the blood of healthy animals, but caused a rapid improvement in the number of blood-corpuscles and the amount of hemoglobin in the anemic animals. This was, however, of short duration. The active part of the bone-marrow was extracted by water, and Fowler thinks that it is a deuteroproteose.

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[1] Zeit. f. Ohren., Bd. xxxv, Hefte 1 und 2.

[2] Lancet, April 28, 1900.

[3] Scottish M. and S. Jour., Sept., 1899.

Burghart[1] used the **extract of spleen and of thymus gland** in various cases of anemia, among which were a case of pernicious anemia and some instances of chlorosis. Some improvement was observed in one case of secondary anemia; otherwise the results were not striking.

A. C. Coles[2] reports a series of blood examinations in 2 cases of pernicious anemia. The rapidity of increase of the red blood-corpuscles in both cases was notable. In the first the corpuscles increased from 1,642,500 to 4,075,000 in a month. In the other case within 2 months the corpuscles increased **from 666,666 to** about **4,500,000,** and the hemoglobin in the same time from 14% to 50%. Ultimately, however, all treatment failed. The best results were obtained from arsenic. The leukocytes were usually found reduced below the normal.

Gautier[3] has frequently observed disturbances of the stomach in the use of **cacodylic preparations** by the mouth; he has also noticed that these preparations at times cause evidence of disturbance in the kidneys, if these organs are not entirely normal. If they are taken over a prolonged period, albuminuria may result. He believes that both these effects, as well as the garlicky odor of the breath, may be avoided by giving hypodermically the cacodylate of soda dissolved in distilled water containing a small amount of phenic alcohol.

P. G. Lodge,[4] in reporting upon the use of **oxygen inhalations** in a number of cases, describes one of severe anemia in which there was a lemon-yellow tint of the skin, the red cells were much deformed and were reduced to 2,000,000, and the hemoglobin was 30%. The first administration of oxygen, which was prolonged for 5 minutes, caused violent respiratory excitement. Subsequent briefer administrations caused much improvement, and within 4 months the red cells increased to 4,000,000 and the hemoglobin to about normal. Later the patient became entirely well.

## LEUKEMIA.

**Etiology and Pathology.**—Minkowski[5] insists that too much attention has been given to the morphology of the blood in studying leukemia, and that both **clinical observations and chemical studies** are likely to prove more valuable than simple morphologic investigation. Chemical investigations show that the chief elements of the leukocytes are nucleinic acid and protamin, which combine with albumins to form nuclein and histon; the lymphocytes are composed almost entirely of nucleohiston, and lymphocytes contain much more nucleinic acid than do the polymorphonuclear leukocytes. The staining properties of the leukocytes are due purely to microchemical reactions with the chemical bodies mentioned. All the variations in the staining reactions are due to the fact that nucleinic acid is polybasic, and at times takes acid and at other times takes basic combinations. Minkowski states positively that uric acid can not be considered to be solely an end-product of the reduction of the nuclein in the leukocytes; it may quite as rationally be considered the result of increased cell function, and is very often the product of the nucleins contained in the food ingested. There is no direct parallelism to be found constantly between the circulating leukocytes and the amount of uric acid excreted. As to the nature of the various forms of leukemia, he considers that changes in the blood in the different forms are due rather to the character which the disease assumes in the organs affected than to the original situation of the disease. He divides leukemia into 3 main classes—genuine general leukemia, acute leukemia, and chronic lymphemia. The first is the ordinary form, which is commonly termed lienal; this gives a polymorphous blood picture, and is probably the result of infection. Acute leukemia is even more probably of infectious nature; the blood changes consist chiefly in increase of the mononuclear elements, the enlargement of the spleen and lymphatic glands is usually only slight, but change in the bone-marrow is practically always found. Chronic lymphemia is closely related to malignant lymphoma and sarcoma; it shows swelling of the lymphatic glands with variable condition of the spleen. Microscopically, one may usually find decided changes in the spleen and bone-marrow, and local leukemic infiltrations in other organs, which are apparently hyperplastic growths. The lymphocytes are always increased. There are many cases which do not correspond well to any one of these 3 classes. As to the importance of leukocytosis, Minkowski does not consider its clinical value very great. The value of the presence or absence of polymorphonuclear leukocytosis as a point in the diagnosis or prognosis of infectious processes is uncertain. Eosinophilia is found in a number of conditions, and is not distinctive of any one disease. Attempts to produce leukocytosis in order to influence the course of infections have been of doubtful value, and alterations in the number of leukocytes in leukemia are not always attended by corresponding changes in the progress of the disease.

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[1] Deut. med. Woch., Sept. 14, 1899.

[2] Brit. Med. Jour., Mar. 31, 1900.

[3] Bull. Acad. de méd., Oct. 21, 1899.

[4] Lancet, April 7, 1900.

[5] Verhandl. des XVII Congress. f. innere Med., 1899.

A. E. Taylor[1] contributes a notable article on leukemia based upon studies of the blood and tissues as well as elaborate investigations into the chemical relations in 16 cases of this disease. No more thorough study of the disease in all its relations has been published. The details of the examinations of the individual cases are first given, and the characteristics of the blood then summarized, with a consideration of the known facts regarding the alterations seen in this disease. Reduction in the number of red corpuscles was present in all of his cases; none of them presenting more than 4,000,000 cells per cubic millimeter, and 3 of them showing under 1,500,000, the lowest being 800,000. In cases in which apparent cure had occurred the oligocythemia persisted. The author does not decide upon the cause of the oligocythemia, but discusses various theories. The red cells, as a rule, contain less than the normal amount of coloring-matter, and incidentally the author takes occasion to state that his own accurate chemical studies have not indicated a high percentage of coloring-matter in true pernicious anemia. His view is that the high color-index to which attention has been so often called is a visual rather than an actual condition. He found polychromatophilia in a certain proportion of cases, and 2 of his cases gave Bremer's diabetic test, though no sugar was present in the urine and the glycogen reaction of the blood was not increased. Poikilocytosis was always present, though only to a slight degree in 3. Nucleated red cells were found in all but 1 of the 16 cases; 10 presented less than 10,000 nucleated red cells; 3, less than 20,000; and 2, from 60,000 to 70,000 per cubic millimeter. One of the first effects of arsenical treatment was a reduction in the number of nucleated red cells when the number had been considerable before the treatment. The majority of the cells were normoblasts, under which designation he includes poikiloblasts. Turning to the questions of the leukocytes, he found ameboid movement less than normal in the polymorphonuclear cells, almost uniformly absent in the large mononuclear cells, entirely wanting in the lymphocytes, but always present in slight degree in the myelocytes. In the myelogenous or leukocytic cases the polymorphonuclear cells were increased in number in all cases, though the relative percentage was reduced. In the lymphatic cases the numbers were not above the normal maximum figure in the majority. The size of the polymorphonuclear cells and their characteristics varied greatly, and the staining reactions, numbers, and even presence of granulations were subject to variations. The oxyphilic polymorphonuclear cells (eosinophiles) were increased in 9 of the leukocytic cases. In 2 the number was normal. In the lymphatic cases 1 showed no eosinophile cells, nor did the blood in this case show any sort of granulation. In the remaining lymphatic cases increase of eosinophiles was exceptional. The nongranular mononuclear leukocytes were always present to excess in leukocytic cases. Their number in lymphatic cases was variable; in 2 they were wanting. Oxyphilic mononuclear cells were present in all cases of leukocytic leukemia. They were absent in 4 of the lymphatic cases. The lymphocytes were present in normal numbers in 7 cases of leukocytic leukemia and increased in the others. In 4 of the 5 lymphatic cases, with one exception, lymphocytosis was extreme. Mast-cells were absent in 2 cases of leukocytic leukemia, but present in marked excess in the other 9. They were observed in but 1 of the lymphatic cases. Myelocytes were always found in leukocytic cases, the number being as low as 1650 per cubic millimeter in one instance, though in this case 4175 oxyphilic mononuclear cells, which are also marrow-cells, were observed. The remaining leukocytic cases presented above 18,000 per cubic millimeter; sometimes very much above this number. In 4 of the cases of lymphatic leukemia no myelocytes were present. In one acute case 20,275 were found. In all his examinations karyokinesis in a white blood-corpuscle was found in but one case. Various forms of nuclear and protoplasmic degenerations were seen. Charcot-Leyden crystals were never seen in the fresh blood nor in properly prepared specimens. The clinical studies of the disease can not be adequately reviewed in this place nor the pathologic investigations. The author looks upon leukemia as of two varieties—a leukocytic or myelogenous form, which he regards as a peculiar type of leukocytosis, possibly dependent upon some infection of doubtful nature; and a lymphatic variety, which is quite distinct in many particulars. The latter also may be a leukocytosis. The two varieties may be combined from the start or in the later stages. The evidences that have been adduced in favor of the view that leukemia is a condition allied to neoplasms are referred to, but the author is not inclined to regard these as adequate. He cites the rapidly destructive and seemingly infectious character of many cases of acute leukemia; the differences between the hyperplasia of the bone-marrow and lymph-glands in leukemia and the hyperplasia of neoplasms; the absence of pathologic mitoses in leukemia; the clinical and pathologic course of the disease and the occasional favorable result of treatment. On the other hand, the origin of leukemia by leukocytosis seems to agree with known facts regarding leukocytosis and the pathology of leukemia. Those who insist upon the neoplastic theory regard the lesions of the fixed organs as primary and the blood condition as the consequence of overproduction of cells; while those of the opposed school maintain the chemotactic origin of the leukocytosis, and view the lesions of the fixed organs as the responsive hyperplasia occasioned by the increased demand. As one evidence in favor of this view he cites the close resemblance of the bone-marrow in dogs in which long-continued leukocytosis has been maintained by injections of albumoses to the marrow of leukemia. His own experiments in this direction are very suggestive. [While we have for a long time recognized the merit of the view that leukemia is possibly an infectious disease, we have never abandoned the older thought that it is allied to neoplasms. There is force in the author's statements in favor of the infectious theory, but it must be remembered that, precisely in the pathology of the lymphatic structures, the distinctions between infectious and neoplastic processes are vague and ill defined.]

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[1] Contributions from the Wm. Pepper Laboratory of Clin. Med., Phila., 1900.

W. Türk [1] believes that the objects which Löwit considered to be **protozoa** in leukemic blood were merely artefacts consisting of **granules from mast-cells.** Löwit [2] replies to this criticism. [Our own opinion coincides with that of Türk to the extent that we regard Löwit's "protozoa" as artefacts or products of cellular degeneration.]

Lipowski,[3] while admitting that the conditions of the blood in leukemia are more distinctive than they are in any other disease, and that the diagnosis can usually be made from the examination of the blood, reports a case to show that this is not always true. This was an instance of carcinoma of the intestine in which, with marked reduction of the hemoglobin and red cells, there was an increase in the number of white cells, so that the proportion of whites and reds was 1 : 18. Nucleated red cells and myeloblasts were present, and had not a tumor been palpable, he considers that the only proper diagnosis would have been leukemia, since the whole glandular apparatus showed swelling. The explanation of this condition was probably irritation of the bone-marrow by metastasis, but such a condition would not have been suspected had not a primary tumor been found. He insists that the **effect of tumors upon the blood** must be held in mind in conditions in which the blood is notably altered. He directs attention to Nothnagel's case in which a true lymphatic leukemia was caused by metastasis of lymphosarcoma to the bone-marrow. The **effect of intoxication upon the blood** is exemplified by a case which he reports in which there was evidently a carcinoma of the liver. There was only moderate change in the red cells, with pronounced reduction of the hemoglobin, while the white cells were increased to the proportion, as compared with the reds, of 1 : 27. A striking fact in the case was that the white cells consisted purely of neutrophiles; not a single lymphocyte or eosinophile cell could be found. Only very late in the course of the disease did a very few lymphocytes and some large mononuclears appear in the blood, but eosinophiles were never found. The explanation appeared to be that chemotactic substances produced an increase in the neutrophiles; but the absence of the other leukocytes must remain unexplained. That a chemotactic action caused the condition was shown by the fact that examination of the slightly enlarged spleen showed numerous lymphocytes, large numbers of nucleated cells, and especially large numbers of polymorphonuclear erythrocytes, and in the bone-marrow all forms of cells were found.

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[1] Wien. klin. Woch., Mar. 29, 1900.

[2] Wien. klin. Woch., April 5, 1900.

[3] Deut. med. Woch., May 24, 1900.

**Symptomatology.**—G. Dock [1] reports **15 cases** of chronic leukemia which he has seen in hospital and mentions 5 others observed in private practice. The proportion of hospital cases of leukemia to other cases was 11.3 : 10,000. This is high as compared with statistics from abroad, but there was no selection of cases, and Dock thinks that leukemia is probably more common in this part of the world than abroad. He has seen but one case of acute leukemia, while Fraenkel saw 12 to 5 of chronic leukemia. Of the whole number of Dock's cases, 11 occurred in men and 9 in women. The average age was 41.6 years; the youngest patient, 27 years. In 5 cases there was a history of chills and fever. There was practically no other etiologic factor of importance. He was unable to discover the protozoa described by Löwit. Dock particularly directs attention to the fact that in only 4 of the 15 hospital cases was the diagnosis suspected or made before the patient was seen at the hospital. This is due either to carelessness in physical examination or to lack of examination of the blood. He particularly notes that many persons think that leukemia is not present if there is no pallor, while pallor is very likely to be absent in leukemia. The red corpuscles averaged in his cases about 3,000,000; the hemoglobin, 56 %. Thirteen of the patients had dyspnea as an early symptom and 10 had edema; in 7 of the latter there was no cardiac or kidney disease to explain the edema.

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[1] Phila. Med. Jour., Mar. 31, 1900.

He thinks that in 7 a history of chills and in 4 a story of eye symptoms should have given rise to an investigation which would have shown the nature of the disease. In only 4 of the 15 cases were the lymph-glands enlarged, and in only 2 was this enlargement distinct. Discussing the diagnosis of enlargement of the spleen, he notes that in 1 case the spleen showed no notch. He directs attention to a history of the primary situation of the enlargement, the sharpness of the edge of the spleen, and the lack of marked fullness in the lumbar space, early in the case at any rate, as of marked importance in excluding enlargement of the kidney. The number of leukocytes was above 200,000 in 14 of the 15 cases; the highest count was 960,000. In this case there were 1,816,000 red cells. The condition of the red blood-cells is believed to be a valuable guide in the recognition of the stage of the case. In 14 cases the duration of the disease when the patients came under observation was fairly well determined, averaging about 8⅓ months.

T. McCrae [1] reports a case of splenomyelogenous leukemia in which there was a marked splenic tumor in June, 1898, with 584,000 white corpuscles, of which 23 % were myelocytes. Three months later there were **no myelocytes and no splenic tumor**; the blood count was practically normal, except for some reduction of the hemoglobin and red cells. The leukocytosis, myelocytosis, and splenic tumor reappeared, and disappeared once more with treatment under arsenic. The man ultimately died after passing from observation, death being said to have been caused by cerebral hemorrhage. McCrae has been unable to find any other case reported in which there was a disappearance of the splenic tumor and of the myelocytes and recovery of an apparently normal blood count.

O. Lerch [2] reports a case of leukemia of mixed type in which a remarkable blood examination was reported, it being stated that the red cells numbered 2,020,000, while the **white cells were 3,570,000.** The man had great enlargement of the spleen, while the liver was normal in size. The blood contained a large number of myelocytes, but no differential count is reported.

Lion [3] reported to the Medical Society of the Hospitals 3 cases of leukemia in which the increase in leukocytes was practically **exclusive of the mononuclear elements.** These leukocytes constituted over 99 % of the whole number, and the large mononuclears were most markedly increased. The cases were chronic, 2 of them having a course of 3 years and 5 years respectively. This class of cases, therefore, differs from the form in which the lymphocytes are increased. Vincent described a similar case, and Widal stated in discussion that he had never seen the sporozoa of Löwit, and had never been able to isolate bacteria from the blood or tissues of patients with leukemia. He considered the large general increase of leukocytes to be evidence against the parasitic nature of the disease, because infections cause an increase of the polymorphonuclear neutrophiles, while in myelogenous leukemia these cells, although increased, are usually present in relatively small numbers, and sometimes are almost the only cells present.

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[1] Brit. Med. Jour., Mar. 31, 1900.

[2] New Orl. M. and S. Jour., July, 1900.

[3] Semaine méd., Mar. 11, 1900.

Widal and P. Merklen [1] report their observation of lymphatic leukemia in a man of 56 of weak constitution. The blood contained lymphocytes almost to the exclusion of other forms of white blood-cells. The autopsy showed the spleen to be very much enlarged and the bone-marrow transformed into a tissue which was composed almost exclusively of lymphocytes. The tissue of the spleen was also almost entirely lymphocytic. Widal agrees with Lion in thinking that there are two types of leukemia in which mononuclear elements predominate: one is the lymphocytic form; in the other the large mononuclear cells are present in greatest number.

A. Petit and E. Weil [2] describe a case in which there was marked enlargement of the lymphatic glands and in which 99% of the leukocytes were mononuclear forms. The case ran its course in about a year. Microscopically, the various organs examined showed collections of lymphocytic tissue.

E. Kreibich [3] describes a case of lymphatic leukemia with **leukemic infiltrations of the skin,** the latter appearing as masses varying in size from that of a pigeon's egg to that of a hen's egg. These were situated chiefly about the head and neck, and were of a reddish color, were somewhat tender, and were covered by glossy skin. Histologic examination showed that the tumors arose in the deeper layers of the skin and consisted of infiltration of mononuclear leukocytes. There were some minute collections similar to these in the apparently normal skin.

W. Murrell and W. Spencer [4] describe the case of a girl of 19 who had marked anemia and who showed extensive **ascites.** The patient had become much reduced, and consequently operation was undertaken, and about 4 quarts of blood-stained fluid were removed and the drainage-tube was left in. The fluid reaccumulated, however. A collateral circulation was established by suturing a portion of the omentum to the abdominal wall. The superficial veins subsequently enlarged and the patient gained largely in general health. The condition was thought to have been a lymphatic leukemia.

**Acute Leukemia.**—H. Reimann [5] describes a case of acute leukemia in a girl 9 years of age. There were indefinite symptoms in the early period of the disease, with hemorrhages from the nose and purpuric skin eruptions. A leukocytosis developed and the spleen, liver, and lymph-glands became enlarged. There was some increase of the eosinophiles, and at first an increase of the polymorphonuclear cells, while in the latter part of the disease the lymphocytes became augmented. There was fever, and late in the attack there was general pigmentation of the skin. The disease lasted 6 weeks in all. In the latter part of the attack the spleen and lymph-glands decreased in size, and at the same time the mononuclear leukocytes increased in number. At a count just before death the red cells were reduced to 450,000, and the white cells increased to 200,000. The hemoglobin is recorded as reduced to 8%. The necropsy showed as the most notable point a decided **enlargement of the thymus gland.**

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[1] Gaz. des Hôp., Mar. 16, 1900.

[2] Gaz. des Hôp., Mar. 30, 1900.

[3] Arch. f. Dermat. u. Syph., Bd. XLVII, S. 185.

[4] Lancet, June 16, 1900.

[5] Wien. klin. Woch., Sept. 28, 1899.

W. N. Bradley[1] reports a case of **acute lymphemia** which was fatal within 8 weeks. It occurred in a boy of 8, and was associated with enlargement of the glands, some fever, dyspnea, and ultimately marked splenic enlargement. The red corpuscles were reduced to below 2,000,000, the whites increased to 85,000; 59% of the latter were small lymphocytes, while the polymorphonuclears were greatly decreased.

E. Hirtz and M. Labbé[2] report a case of subacute leukemia in a man of 21. The disease ran its course in about 9 weeks. The etiology was entirely unknown, though there was a **nasal infection,** and perhaps this had caused the disease. The condition was probably chronic leukemia of mild degree, upon the basis of which a fatal septicemia developed. Bacterial emboli were found in the spleen and bone-marrow. The blood showed marked increase of the lymphocytes.

**Complications.**—E. Körmöczi[3] describes a case of leukemia in which **suppuration** occurred **in the antrum of Highmore.** The man had fever until the last few days of life, when the temperature became subnormal. About the same time the leukocytes decreased from 20,000 to as low as 1000 per cubic millimeter, and the red cells decreased from 1,400,000 to below 400,000. The reduction of the leukocytes was attributed to the effect of bacterial poisons upon the blood-producing organs, the organs becoming unable to elaborate either red or white corpuscles; it was not thought to be due to chemotaxis, since ordinarily infections increase the polymorphonuclear cells and decrease the eosinophiles, while in this case there was no increase of the polymorphonuclears and the whole number of leukocytes was decreased. Körmöczi considers that infectious diseases may cause a reduction in the leukocytes owing to a destructive action of the bacterial poisons upon the tissues, or the action may be chemotactic, resulting in increase of the polymorphonuclears and decrease of the others. The red cells are usually but little affected, though they may be very greatly decreased in cases in which the destructive action of the toxins is predominant.

P. Weber[4] reported a case of leukemia in a man of 31 in which there appeared 6 months before death attacks of headache, vertigo, and vomiting, and the man shortly afterward became entirely deaf. The condition was considered **Ménière's disease.** The postmortem examination of the ears showed that a portion of the scala tympani of the one side examined, and the perilymphatic spaces of the semicircular canals on both sides, were filled with newly formed fibroid and bony tissue. There were no obvious changes in the nerve-trunks.

H. Schroeder[5] describes a case of leukemia in a woman of 25 which apparently began in the fifth **pregnancy.** She was observed in the sixth pregnancy, at which time the white cells were very largely increased. Smears showed the usual characteristics of leukemia. The spleen was much enlarged. The disease was advancing rapidly, and labor was artificially induced. The woman had some fever afterward, but rapidly improved, and her general condition became fairly good, though the blood still showed leukemic characteristics. The fetus presented no evidences of leukemia.

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[1] N. Y. Med. Jour., Dec. 23, 1899.

[2] Gaz. des Hôp., Mar. 16, 1900.

[3] Deut. med. Woch., Nov. 23, 1899.

[4] Brit. Med. Jour., Mar. 3, 1900, p. 510.

[5] Arch. f. Gynaek., Bd. LVII, No. 1.

## SPLENIC ANEMIA.

[There has been a tendency to revive this term, which for a number of years had almost wholly disappeared from literature. Most authors admit that there is not a definite entity to which the name is applicable, but that the term is a convenient one to designate a clinical picture, probably produced by a variety of pathologic conditions.]

W. Osler [1] describes 15 cases under the heading of splenic anemia, using this term as an indication of the class of cases in which the chief signs are **marked splenic enlargement with anemia, but without involvement of the lymphatic system.** In the cases reported the enlargement of the spleen apparently preceded the anemia. The splenic enlargement caused no inconvenience, although great in all cases. In 8 of the cases reported there was hemorrhage from the stomach, and in 7 instances these symptoms caused the patient to consult a physician. Ascites occurred in 3 cases; in one patient upon whom autopsy was made the liver was found free from cirrhosis. The appearance was sometimes that of a slight anemia; sometimes the pallor was almost as intense as that seen in progressive anemia. There was marked bronzing of the skin in some of the cases; in one it was extremely pronounced. The red blood-cells were only moderately reduced, as a rule, but the hemoglobin was always found to be relatively very low. The leukocytes were normal or reduced in number, frequently the latter.

B. W. Sippy [2] reports a case of splenic anemia in a man of 45, the disease appearing with general weakness, disturbance of the gastro-intestinal tract, irregular fever, enlargement of the spleen, which advanced to a pronounced degree, moderate enlargement of the liver, and ascites. The red blood-cells were reduced to 1,740,000; the whites were 5214; the hemoglobin was 30%. There were no changes in the relative number of the different forms of leukocytes, and nucleated red cells were absent. The man became progressively worse, and died in a marasmic condition. Autopsy showed marked collections of lymphoid elements in the spleen as well as in the liver and lymphatic glands, and in all these organs there was some increase of the fibrous tissue. The bone-marrow showed chiefly collections of lymphoid cells with connective-tissue increase and the presence of nucleated blood-corpuscles. There were polypoid growths of the stomach, which consisted of proliferation of the gland structures. During life the spleen was punctured, and culture-tubes and animals were inoculated. No result was obtained, except one culture of Staphylococcus aureus; this organism was probably the result of an inflammation of the ear which existed at the time. Cultures made at the autopsy were also negative except for the presence of the colon bacillus and staphylococci. The injection of splenic emulsion caused rapid death in 2 rabbits. Cultures from the blood proved negative. It was thought that the **spleen contained some abnormal toxic substance.** Sippy believes that in splenic anemia the splenic enlargement is probably primary, and that it may perhaps produce the symptoms in a manner similar to that in which the symptoms in exophthalmic goiter are probably caused; enlargement of the spleen and derangement of its function perhaps result in the production of toxic substances which cause nutritional disturbances. In treatment he strongly recommends operation, which should be undertaken as early as possible. He refers to 7 cases which he has collected in which operation was undertaken with only 2 deaths; the other 5 recovered entirely. [The diagnosis in certain of these cases is, however, very questionable, and most of them were reported before the study of the blood was sufficiently far advanced to make a diagnosis possible.]

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[1] Am. Jour. Med. Sci., Jan., 1900.

[2] Am. Jour. Med. Sci., Oct., 1899.

W. Türk [1] describes a case which was observed in a man of 56 who had a history of syphilis. He had marked anemia and irregular slight fever, with great enlargement of the spleen and liver. There was no leukocytosis. The case was called splenic pseudoleukemia. The man died, and autopsy disclosed **lymphosarcoma** of the retroperitoneal glands, of the spleen, of the mediastinal glands, and of some of the other glands. The spleen also showed chronic engorgement. The tumors consisted of mononuclear leukocytes with very little reticulum. The author goes over the question of lymphatic tumors and hyperplasias of the lymph-glands, discussing at the same time pseudoleukemia and leukemia. He believes that all these cases might be put under the following heads: first, partial lymphomatosis, in which there are local enlargements that are not destructive; second, universal lymphomatosis with general lymphoma; third, lymphomatosis destruens, in which there are destructive forms of lymphoma; and, fourth, lymphosarcomatosis arising locally and causing metastasis. The third and fourth forms are much alike. He does not believe that the term pseudoleukemia as it is commonly used indicates a single morbid process. He thinks that all these cases had better be classed under **lymphomatosis,** and distinguished by the terms he has suggested.

N. Dalton [2] reported to the Clinical Society of London a case of anemia with enlargement of the spleen which clinically presented the characteristics commonly described under the name of splenic anemia. The patient died suddenly, and the postmortem showed that the stomach was much distended, was strangulated, and that ultimately there had been a perforation, the strangulation being due to abnormal position of the stomach, so that when it became distended and rotated upon itself, both the cardiac and pyloric orifices were much constricted. The enlargement of the spleen was thought to be due to **obstruction of the splenic**

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[1] Wien. klin. Woch., Oct. 5, 1899.

[2] Lancet, Nov. 18, 1899, p. 1371.

**vein.** Dalton considers that there is no proof that the splenomegaly may be considered a pathologic entity. He thinks that there is no necessity for giving the name splenic anemia to a condition which has no distinctive characteristics separating it from other diseases. P. Kidd, in discussion, also expressed doubt about the existence of a splenic anemia, and insisted that he had repeatedly observed instances of cirrhosis of the liver with great enlargement of the spleen and marked anemia, and he thought that probably such cases were sometimes mistaken for so-called splenic anemia.

H. A. Hare [1] contributes a supplementary report of a case previously reported which was considered to be splenic anemia, or Banti's disease. The chief interest in this report is that while the man had formerly been free from any such symptoms, he suddenly developed severe glycosuria with polyuria and other symptoms of **diabetes**—a complication of splenic anemia which seems to have been previously undescribed. The man disappeared from observation. He had shown signs of rapid improvement in his blood condition under the use of arsenic.

## THE HEMORRHAGIC DISEASES.

Mannaberg and Donath [2] investigated 3 cases of **paroxysmal hemoglobinuria.** A fresh attack seemed to be readily produced by having the patients immerse their feet in iced water for various periods. Exposures for varying times always brought on attacks quite similar to those produced by natural exposure. Bandages placed tightly about the arm until cyanosis was produced caused the appearance of hemoglobin in the blood-serum. When blood was withdrawn and shaken, it was found that, as compared with control cases, separation of the hemoglobin occurred much more rapidly. They suspected that a hemolytic ferment might be present in the blood in these cases, and found that when blood from a normal person was added to blood in which hemolysis had occurred, there was distinct degeneration of the corpuscles.

F. Schuffer [3] notes that the theories concerning the **origin of hemoglobinuria** are that the destruction of the corpuscles takes place in the kidneys; and, on the other hand, that it occurs elsewhere, and that the kidneys merely excrete the hemoglobin. He investigated the question by injections of distilled water into the renal veins or of glycerin subcutaneously. His most important result was that the amount of hemoglobin found free in the circulating blood was less than that in the urine. This and other factors led him to conclude that the hemoglobin was abstracted from the red corpuscles by the kidneys, and he believes that the kidneys are often the primary seat of the trouble.

P. S. Roy [4] reports a case of paroxysmal hemoglobinuria which occurred in a woman of 60 who was fatigued after sight-seeing. She had previously had **Raynaud's disease,** and subsequent to this attack had **multiple arthritis.**

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[1] Jour. Am. Med. Assoc., Dec. 30, 1899.

[2] Deut. Arch. f. klin. Med., Bd. LXIII, Hefte 3 u. 4.

[3] Il Policlinico, Aug. 30, 1899.

[4] Phila. Med. Jour., Sept. 21, 1899.

## PURPURA.

D. B. Lees [1] reports a case of subacute **rheumatism** which was complicated by purpura in the early part of the attack. There was also severe **endocarditis.** Some of the bullæ ruptured and left raw surfaces. Blood cultures showed **streptococci,** and **staphylococci** were found in the fluid of the bullæ. There was no leukocytosis. The girl recovered from the pupura, but afterward died from cardiac failure.

Mayer [2] describes the case of a boy with pneumonia in which he gave **turpentine** for the purpose of exciting leukocytosis. He gave 3 drops of a mixture composed of 3 gm. of turpentine and 5 gm. of cinnamon. Crisis occurred, but was followed by severe bleeding from the nose and the development of purpuric spots over the whole body. Death occurred from heart weakness. Mayer attributes the purpura to the turpentine.

**Treatment.**—Arcangeli [3] gave subcutaneous injections of a 2% solution of **gelatin** in normal salt solution in 2 cases of purpura hæmorrhagica which occurred in girls 13 and 10 years of age. The largest dose used was 20 cc. The hemorrhages ceased entirely after several injections.

## HEMOPHILIA.

W. R. Steiner [4] reports a case of **hemophilia in a negro** girl of 14, which he states is the second case reported as occurring in the negro race. The patient's grandmother was a bleeder, and had 14 children; 7 of these whose histories were known to the mother of the patient were bleeders, and all were dead but one. The mother had had epistaxis until she was 16. The patient's brothers tended to bleed freely from slight cuts. The girl whose case is reported had been a bleeder since early childhood. Steiner draws attention to the fact that this family, as is frequently observed with bleeders, showed marked fertility; that many of the children died young, also a common observation; while, contrary to the usual experience, both males and females seem to have been bleeders. As usual, the males preponderated over the females in the family, but the bleeding tendency was in every case transmitted through the females.

Heymann [5] used subcutaneous injections of **gelatin** solution in the case of a patient with hemophilia. There had been prolonged bleeding after removal of the tonsils. The bleeding ceased, but later recurred. It was controlled with the gelatin, and did not again recur. Other treatment had been ineffectual. J. B. Nichols [6] reports a case in which there was a family history of hemophilia and prolonged bleeding after a wound of the wrist. It did not cease after the application of the tourniquet, but was rapidly controlled by pouring gelatin into the wound.

G. W. Wagner [7] describes 3 cases of hemophilia that were successfully treated by leaving the **bleeding points exposed to the air,** in one case also using hot water. Wagner gives a general description of the disease.

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[1] Lancet, Oct. 28, 1899.

[2] Zeit. f. Medicinalbeamte, No. 2, 1900.

[3] Semaine méd., 1899.

[4] Johns Hopkins Hosp. Bull., Feb., 1900.

[5] Münch. med. Woch., Aug. 27, 1899.

[6] Med. News, Dec. 21, 1899.

[7] Physician and Surgeon, Sept., 1899.

## SCORBUTUS.

F. G. Jackson and V. Harley [1] have inquired into the experience of Nansen and other explorers, and also have done some experimental work concerning the **causation of scurvy.** In the latter work monkeys were fed on a diet composed of rice and a considerable quantity of meat, the first series being given fresh meat, the second tainted meat, and the third series tainted meat with a banana or an apple. The result was apparently to show distinctly that the monkeys had no symptoms of scurvy when fed on fresh meat; but that when fed on tainted meat, both when given fruit and when this was withheld, they showed pronounced symptoms of scurvy and the blood had the characteristics found in this disease. The authors have also observed in the records of Nansen and others quoted that living on fresh meat did not cause scurvy even though no fruit was taken, but it has frequently been noticed that taking tainted meat will cause scurvy, and that this will not be controlled by eating fruit. They decide that the disease is caused by poisoning from tainted or canned meat.

## DISEASES OF THE SPLEEN AND LYMPHATIC GLANDS.

Reich,[2] as a result of experimental work, decides that the spleen undoubtedly has a **hemolytic function.** He observed what was apparently direct metamorphosis of pigment from the red corpuscles. In the frog's spleen the protoplasm of the cells showed cavities and defects about the edges of the cells, and he subsequently observed round bodies which reacted to tests for hemosiderin. The red corpuscles subsequently became largely filled with pigment granules, and the nuclei lost their structure and showed karyolytic and karyorrhectic changes.

Kraebel [3] reports a case of **extirpation of the spleen** which was undertaken after rupture of this organ. At the time the patient left the hospital, a month after the operation, his hemoglobin was 80 % and he was apparently in good condition. There was slight enlargement of the lymphatic glands, but no change in the thyroid.

M. Laub [4] reports 4 cases which, he considers, come under the heading of **status thymicus.** They were all fatal, and occurred in male patients between 17 and 24 years of age. They were all brought to the hospital in an unconscious condition; in all of them the spleen was discovered to be enlarged during life; and at postmortem the thymus was found to be enlarged and there was enlargement of the spleen and lymph-glands. The author considers that with a history of sudden unconsciousness without any evident cause, coming on in apparently perfect health and without evidence of cerebral disease, a diagnosis of status thymicus is very probably correct if the spleen is enlarged or if enlarged follicles may be found at the base of the tongue or in the nasopharynx.

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[1] Lancet, April 28, 1900.

[2] Fortschr. d. Med., 1899, p. 361.

[3] Deut. med. Woch., Sept. 7, 1899.

[4] Wien. klin. Woch., Nov. 12, 1899.

## DISEASES OF THE CIRCULATORY ORGANS.

### METHODS OF EXAMINATION.

W. Coleman[1] describes a **chart** consisting chiefly of concentric circles divided so that the time of occurrence of the heart-sounds may be recorded. Thus the relation of the murmurs to the various phases of the heart-cycle may be determined in difficult cases.

A. Abrams,[2] as a means of **measuring the intensity of the heart-tones,** uses rods of partly vulcanized rubber, which are placed between the stethoscope and the chest. The length of the rods is increased until the heart-tones become inaudible, and the intensity of the tones is thus approximately determined.

R. Van Santvoort,[3] in discussing the use of the **sphygmograph,** states that he considers that this instrument provides a fairly approximate method for estimating the amount of arterial tension. He thinks that the variations seen in sphygmograms in cases of aortic regurgitation are not evidences of the unreliability of the instrument, but of different conditions of the vessels and of different degrees of regurgitation. He recommends that a series of sphygmograms be taken from each case examined, and that the sphygmogram which seems to represent best the condition in the case should be chosen and used in diagnosis and treatment. [Sphygmograms should be made according to a uniform method, the place of application of the pressure button being always the same, and pressure upon the head or shaft of the radius being carefully avoided. Lack of attention to such details as this accounts for some of the variable results reported.]

### GENERAL CONSIDERATIONS CONCERNING DISEASES OF THE HEART.

A. Morrison,[4] after an extended clinical study of the **causes of the first sound** of the heart, decides that the chief factor in the production of this sound is the vibration of the blood within the cavities containing it, though the surrounding vibratory tissues aid in its production. He was led to this conclusion by observing that in severe congenital or other lesions the first sound is still good as long as the division between the two columns of blood is fairly well maintained; and that, on the contrary, the first sound may be clearly heard when there are extreme valvular lesions which practically may be considered to prevent the valves from making much sound, and when the muscular force of the heart is reduced to zero.

Frank and Voit[5] investigated the question of the occurrence of

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[1] Med. Rec., July 15, 1899.

[2] Med. News, July 8, 1899.

[3] Med. Rec., Feb. 24, 1900.

[4] Lancet, May 19, 1900.

[5] Deut. Arch. f. klin. Med., Feb. 6, 1900.

**asynchronous action of the ventricles** by introducing metal catheters into each ventricle and connecting these with manometers which registered upon drums. They then suffocated the animals or poisoned them with ergot. They could never find any clear evidence of asynchronous action of the ventricles.

K. Doll [1] gives an extensive review of the literature upon the **double apex-beat** of the heart, and reports a case in which an explanation of the double apex-beat seemed evident. Necropsy showed an aneurysmal bulging at the apex of the left ventricle, and Doll believes that the double apex-beat was due to the presence of the aneurysm, its filling producing the first beat, while the second beat resulted from the normal systole. The second beat was coincident with the pulse. It is evident that no one explanation is sufficient for all cases. The two explanations which seem usually most satisfactory are short diastole followed by an excessively rapid systole, thus producing two beats very close to each other, or a lack of synchronous action of the two ventricles, producing the double beat.

Determann [2] reports a series of investigations concerning the **freedom of movement of the heart.** He determined the position of cardiac dullness carefully when persons were in different positions, and confirmed his results by the use of the x-rays. He records a large number of observations. In normal persons he found that in the left-sided position the heart moved as much as 2.5 cm. toward the left and 1 cm. upward; when lying on the right side, it moved 1.5 cm. toward the right, on an average, and 0.5 cm. upward; sometimes the movements to the left were as much as 6.5 cm., those to the right 4 cm. The degree of distention of the stomach caused variations in this, as did high position of the diaphragm, pregnancy, and any disease of the thorax or abdomen which limited the space in the thorax. A very free movement was found in chlorosis as a result of the relaxation or weakness of the vessel walls; the opposite has been observed in arteriosclerosis. The free movement in neurasthenia seemed to be connected with general reduction of the nutrition, and perhaps was due to a lack of fat. Probably the causes of increase in the degree of movement are chiefly relaxation of the tissues, imperfect development of the connective tissues, or perhaps overstrain of the heart, which has overstretched the tissues holding it in place. The symptoms caused by it are chiefly those often observed by normal persons when lying upon the left side, or sometimes when lying upon the right. The pulse becomes frequent, sometimes irregular, and the patients often complain of marked oppression. These symptoms are probably due to mechanical disturbance of the circulation. The reaction to the free movement of the heart is probably proportionate to the sensitiveness of the organs of the individual.

A. Hoffmann [3] reports a further case to show that **distention of the stomach,** and other conditions causing elevation of the diaphragm, may cause some torsion of the heart, so as to increase the lateral dullness, and to give the clinical impression that the heart is dilated. It is important to remember that the lateral dullness of the heart is usually somewhat increased when the heart is displaced upward by any abdominal condition.

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[1] Berl. klin. Woch., Oct. 2 and 9, 1899.

[2] Deut. med. Woch., April 12, 1900.

[3] Deut. med. Woch., May 10, 1900.

T. E. Satterthwaite[1] discusses the **displacement of the viscera** of the thorax and abdomen which results from lateral curvature of the spine. The thoracic organs usually show most marked displacement, since the greatest deformity is in the thoracic region. Satterthwaite has observed cases in which he thought curvature of the spine was the chief cause of disease of the heart and lungs, and some instances in which correction of the curvature caused marked improvement in the conditions of the heart and lungs without other treatment. He advises the use of resistance exercises and of forcible pressure, massage, faradism, and carbonated brine baths, together with nutritious food. Spinal supports have only a palliative effect, and often do harm by preventing the proper degree of muscular exercise.

Placzek[2] has made a study of the **pulsatory foot phenomenon.** He considers that the phenomenon is due chiefly to the compression of the popliteal artery when the knees are crossed, though any position which causes constriction of the artery has much the same effect. He believes that it probably is not a patellar reflex, excited reflexly, as Rosenbach teaches, since it is present in tabetic patients who have lost their knee-jerks, and it is synchronous with the pulse; it is possible, however, that the theory may have some truth. He describes tracings obtained in the study of the foot phenomenon in health and in various forms of cardiac disease. The tracing is more marked than that obtained from the pulse of the wrist, and comparatively its sphygmogram is reversed.

M. Heitler[3] has investigated the relations between the **volume of the heart, the pulse, and the liver,** and the influence of irritation of the liver upon the heart. In arhythmia Heitler observed that the volume of the heart corresponded to the changes in the volume of the pulse, the cardiac dullness enlarging with a weaker pulse and decreasing with a fuller pulse. The liver and splenic dullnesses varied directly in proportion to the cardiac dullness. Percussion of the precordia caused the pulse to become fuller and stronger; percussion of the thorax elsewhere had no influence; percussion of the liver also caused increased strength and fullness of the pulse; percussion of the spleen had no influence upon the pulse. [The practical value of these observations is not very apparent, and it does not seem likely that any series of cases will give uniform results.]

H. Herz[4] discusses **active dilation of the heart.** It has been shown, particularly by Rosenbach, that this is a common clinical condition. It consists in excessive diastole. The heart does not, as was formerly taught, dilate purely passively, but dilation of the cavities is largely an active process, and takes place in such degree as to accommodate the necessary amount of blood for the following systole. In some cases—as, for instance, in the anemias—in which the quality of the blood is poor, a larger supply of blood is demanded. The heart meets this demand by prolonging the diastole in order that it may expel larger quantities of blood. Hyperdiastole may occur also in overexercise, in sexual excess, in obesity, and in abdominal plethora, and occasionally in many other conditions. The clinical characteristics are some widening of the heart dullness; a diffuse, forcible, and rather deliberate cardiac impulse, indicating increased labor of the heart; a full soft pulse that tends to become rapid upon slight excitement; and the presence of varying murmurs of the cardiopulmonary type. Subjective symptoms are either absent or consist of restlessness and irritability, with more or less precordial distress, and perhaps actual pain. Sleep is often disturbed. The diagnosis is chiefly between this condition and passive dilation and hypertrophy. It is discriminated from the latter by noticing the diffuse laboring impulse and the tendency of the symptoms to disappear rapidly. From passive dilation it is diagnosticated chiefly by the full pulse and the absence of signs of cardiac insufficiency. The prognosis is good if the cause can be removed. It is important that the condition should be recognized early, before any more serious change has supervened. [More convincing proof of the possibility of occurrence of active dilation is desirable.]

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[1] N. Y. Med. Jour., Sept. 30, 1899.

[2] Berl. klin. Woch., July 31, 1899.

[3] Wien. klin. Woch., Dec. 28, 1899.

[4] Deut. med. Woch., Feb. 22 and Mar. 1, 1900.

A. Folwell [1] considers dilation of the right conus and pulmonary artery a constant abnormality in **functional cardiac murmurs** in the pulmonary region. This dilation of the conus and pulmonary artery is produced by exertion in debilitated subjects. If the strain is greater, the tricuspid and mitral valves also give way. He explains the primary yielding of the conus and pulmonary artery by considering the ventricle as the half of a cone bisected from apex to base. The firm septum between the ventricles remains strong as long as the left ventricle does not fail. The remaining portion is yielding, particularly along the semicircular border of the base, and the conus will stretch most because the remaining portion is held intact by the fixed inferior vena cava and by the cordæ tendinæ and the papillary muscles. Also, the blood current passes from the apex of the hypothetic cone toward the pulmonary valves, and the conus is therefore distended with blood. Dilation of the conus carries the pulmonary valves upward, and thus leads to relaxation of the pulmonary artery, and hence it is more subject to distention and dilation. [The author's view regarding dilation of the conus corresponds to that of one of the editors (S.). The same opinion has been held by others.]

A. E. Sansom [2] discusses the **effects of influenza** upon the heart and circulation. The joint attacks which occur in influenza he considers nonrheumatic, and rather of the nature of osteo-arthritis, and thinks that they are due primarily to disease of the nervous system, with secondary joint changes. In the heart disturbances of influenza the cause is also an affection of the nervous mechanism. The commonest sequel is tachycardia, which may appear during the attack or a considerable period afterward. There is usually no other change in the heart itself than dilation, which may be only temporary. There are often some symptoms of Graves' disease associated with the tachycardia. It is best treated by weak continuous galvanic currents over the pneumogastrics; by salt water sponging, and proper management of any gastro-intestinal disturbances. Irregularity is also common, and with this there are likewise frequently symptoms of exophthalmic goiter. Bradycardia occurs either as a paroxysmal or as a constant affection, and it is the most dangerous of the three disturbances mentioned, being likely to lead to syncope, which may be fatal. It is difficult to treat, but it sometimes subsides under the use of belladonna. Cardiac pain simulating angina pectoris is quite common, but is rarely dangerous. Sansom has repeatedly seen clinical postmortem evidences of aortitis after influenza, the symptoms being severe pain after slight exercise, sometimes high tension, particularly during the attack of pain, and a dead leaf complexion. Elevated patches are found in the aorta after death, and often reduce the lumen of the coronary artery. The first changes are found in the terminal branches of the nutrient arteries of the aorta, and consist of edema and round-cell infiltration. These cases are not infrequently fatal. They are sometimes helped by the use of iodids.

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[1] Lancet, Nov. 4, 1899.

[2] Lancet, Oct. 21, 1899.

R. Saundby,[1] in discussing the **influenza heart,** directs attention to the fact that functional alterations in the rhythm—usually bradycardia, less often tachycardia—are commonly seen after influenza. In other cases there is definite organic disease, usually dilation. The distinction between the two conditions is not always readily made, and they are often combined. While the change, as a rule, is not severe, yet not infrequently it may become alarming, and the effect of the influenza upon the heart is at times ultimately fatal. Dilated heart after influenza is not common in women in his experience. The most important points in treatment are rest, careful attention to the gastro-intestinal tract, and the use of iron and arsenic, and in some cases of digitalis; but he considers the use of the Nauheim baths more important than all these in cases of dilation.

Roeger[2] discusses **angina with endocarditis.** Heart murmurs were present in 24 of 120 cases of tonsillitis, or in 20%; in 24 cases of herpetic tonsillitis and pharyngitis heart murmurs were heard in 13 instances; in 10 the heart murmurs developed while in the hospital. In 8% of the cases the murmur was permanent, indicating that there had been an actual endocarditis.

F. A. Packard[3] describes 5 cases of **endocarditis** which occurred **with tonsillitis.** In all these cases there was an absence of history of rheumatism, and there was no reason for thinking that the tonsillitis was related to rheumatism. Packard does not think that we are justified in considering the tonsillitis rheumatic in such cases. He believes that probably the tonsillitis is an infection independent of rheumatism, and one that may produce endocarditis or inflammation of the joints, or both.

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[1] Birmingh. Med. Rev., Nov., 1899.

[2] Münch. med. Woch., Feb. 20, 1900.

[3] Am. Jour. Med. Sci., Jan., 1900.

E. Berie[1] describes 2 cases of **malignant endocarditis of rheumatic origin.** The rheumatism was of moderate intensity, but the endocarditis was severe from the first. One patient was a habitual alcoholic, and the other was of very depraved physical condition.

L. La Vastine[2] reports a case of **infectious endocarditis** which occurred during the course of **puerperal fever.** Bacteriologic examination of the blood showed the presence of numerous streptococci with a few staphylococci. The use of antistreptococcic serum was followed by improvement, repeated doses resulting in recovery.

Loeb[3] describes a case of **endocarditis** which came on **after gonorrhea,** and was complicated by joint symptoms. The cardiac symptoms were those commonly seen in endocarditis. The autopsy showed extensive lesions of the aortic valve, and diplococci were found in the valve which had the morphologic appearance of gonococci. Loeb found that gonorrheal endocarditis is more common in women than in men. Altogether, 73 cases have been recorded as due to this cause. The aortic and pulmonary valves are most commonly affected.

R. H. Fox and E. A. Lermitte[4] report a case of infectious endocarditis in which large amounts of **antistreptococcic serum** were used, 15 doses of 10 cc. each being given. There was no result from this treatment. Nuclein was then given without effect. He then used an eclectic preparation containing croton oil, or a similar drug. Improvement occurred for a few days, but the condition soon grew worse, and the patient died. Staphylococcus pyogenes was found in the blood, and there was an acute endocarditis of the aortic and mitral valves, and some pericardial effusion. There was also effusion in the left pleural cavity and in the peritoneum.

E. G. Janeway,[5] in the report of a number of **clinical observations upon heart disease,** mentions two cases of mitral regurgitation which showed disappearance of the murmur years after it was known to have been present. In a third case in which there had been signs of mitral regurgitation death resulted from other causes, and postmortem examination proved that the valves had become entirely normal. In discussing malignant endocarditis he makes two classes—those with murmur and those without murmur, the latter form being particularly difficult to recognize. He considers an eruption of petechiæ of a somewhat nodular character upon the palms and soles as particularly indicative of malignant endocarditis. One case described occurred soon after gonorrhea. There was intermittent fever, and a murmur developed over the heart. There was marked leukocytosis. The man recovered, however, and it was believed to have been a recovery from malignant endocarditis.

Litten[6] discusses a form of **acute fatal endocarditis** which occurs with rheumatism, but is **not of the usual pyemic form** of malignant endocarditis. It differs from the ordinary rheumatic endocarditis in that it does not become stationary and tend to form connective tissue, but shows the signs of a general severe infection with marked involvement of the heart; the temperature persists, but is usually low; the spleen enlarges, there are often hemorrhages in the skin and mucous membranes, acute nephritis is observed frequently, and metastasis occurs. But in the metastases is seen the most marked distinction from ordinary malignant endocarditis: *i. e.*, in the latter the metastases are usually suppurative, while in the form under discussion metastasis produces only infarcts and anemic necrosis.

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[1] Semaine méd., Jan. 21, 1900.

[2] Presse méd., Oct. 4, 1899.

[3] Deut. Arch. f. klin. Med., Bd. LXIII, Hefte 3 u. 4.

[4] Lancet, Nov. 4, 1899.

[5] Med. News, Aug. 26, 1899.

[6] Berl. klin. Woch., July 10 and 17, 1899.

Ebstein,[1] in discussing **malignant endocarditis,** notes that it may follow an acute, a subacute, or a chronic course. It has a variable symptomatology. The early appearance of cerebral symptoms is a bad sign, and the general symptoms are an index to the malignancy of the individual case. A variable fever, tending to run high, and sometimes of the inverted type, is an important sign. Oftentimes endocardial changes can not be demonstrated clinically, and the case must be called merely cryptogenetic septicopyemia. When foci are present elsewhere, it may be impossible to make a diagnosis of endocarditis.

R. Stinzing [2] discusses the relation between **heart disease and epilepsy,** reporting two clinical observations of such an association. After studying the literature he reaches the conclusion that heart disease and epilepsy are, as a rule, related to each other only in an accidental way. Epilepsy at times causes dilation of the heart, but that is usually temporary, and no permanent results follow. Heart disease is probably never the sole cause of epilepsy, but heart disease and arteriosclerosis often make epilepsy worse, probably through circulatory disturbances in the brain. This is particularly notable in senile epilepsy. Oftentimes cardiac medication will have a happy effect on epilepsy. Heart disease often becomes worse during the course of epilepsy, and at times the epileptic attacks take a peculiar form, showing a cardiac aura preceding the attack, and angina pectoris after the attack.

T. Geisler [3] discusses the form of **angina pectoris** that occurs with the climacteric. He attributes it largely to a neurasthenic or hysteric origin, but believes that it may be due to vasomotor conditions, and possibly to spasm of the coronary arteries. He recommends the use of ovarian extract in the treatment.

J. Carslaw [4] describes a peculiar **cardiopulmonary rale** heard in a young man with incipient phthisis. Upon holding the breath after a deep inspiration, there was a peculiar muscular clicking sound, synchronous with the heart-beat, which was heard even at a distance from the patient. It was believed to have been caused by the presence of a cavity near the heart or aorta, the heart or vessels producing the sound by impact upon the walls of the cavity.

R. G. Curtin [5] describes a case of disease of the heart in which a

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[1] Deut. Arch. f. klin. Med., Bd. LIII, p. 217.

[2] Deut. Arch. f. klin. Med., Bd. LVI, S. 241.

[3] Vratsch, 1899, No. 7.

[4] Brit. Med. Jour., July 8, 1899.

[5] Med. News, Sept. 9, 1899.

**curious murmur** synchronous with the heart-sounds was noted, and was still present when the breath was held, becoming louder when the mouth was open and diminishing in intensity when the mouth was closed. He compares the sound to that of a gush of air, and attributes it to rhythmic compression of a distended esophagus by the hypertrophied heart, referring to a similar case of Fulton's in which the autopsy disclosed nothing but hypertrophy of the heart. In another case Curtin discovered a peculiar clicking sound synchronous with the heart action and occurring at the time of respiration. It was present most markedly after exercise, after the ingestion of alcohol, or after indulgence in a full meal. It vanished if the breath was held. He could not explain its causation.

R. Cabot [1] reports a case of **chronic cyanosis without discoverable cause.** It finally ended in cerebral hemorrhage. The patient was a woman of 48, who had experienced repeated collapse with marked cyanosis. The urine contained a little albumin, and there was some increase in both the red and white corpuscles during the attack. The heart seemed to be normal. Marked hemorrhage occurred after slight injuries. She ultimately developed a hemiplegia, and the autopsy showed a small hemorrhage from the middle meningeal arteries, but nothing else except a severe congestion of the organs. Cabot [2] later describes a similar case which occurred in a woman of 49, who was believed to be suffering from lead-poisoning. She had slight dyspnea and marked cyanosis, the blood count and hemoglobin being excessively high. The spleen was somewhat enlarged, there was polyuria, and she had headache and dizziness. No cause for the condition could be found.

C. J. Martin and G. E. Renne,[3] in discussing **cardiac thrombosis,** state their belief that firm white fibrinous clots are formed only antemortem. In making 450 autopsies they found cardiac thrombosis to some extent in 42% of patients. Of these patients, 80% had died of disease of the lungs or from suppurative diseases. In the same class of diseases they observed in other instances that at autopsy the blood was fluid and had lost its coagulability. Diseases frequently associated with either increase or decrease in coagulability of the blood are those in which there is marked disintegration of some of the cellular tissues of the body; and probably some of the changes in coagulability are due to nucleoalbumins, which are known experimentally to produce clotting at times when injected into the circulation, and at other times to cause the blood to lose its coagulability. Also, clotting occurs more readily in places where the tension of carbonic acid is highest, and also where the movements of the fluid in the circulation are most rapid; hence since carbonic acid is present in greater amounts on the right side of the heart, and since here the blood is rapidly churned by the movements of the heart, clotting is likely to occur, and here the products of cell destruction are probably present in greater amount. The threads of fibrin form first in the auricle, advance into the ventricle, and thence into the pulmonary artery. The explanation of the occurrence of clotting at one time and of loss of coagulability at another probably consists in the fact that nucleo-albumins may be split up into the nuclein and the proteid. The nuclein is active in the formation of fibrin; the proteid hinders coagulation. The action of the nuclein occurs first; but ultimately the influence of the proteid histon, particularly if present in large amounts, predominates, and the blood does not coagulate. The authors think that frequently patients with pneumonia die of cardiac thrombosis, and that it is possible that a system of treatment may be developed which will prevent this occurrence.

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[1] Boston M. and S. Jour., Dec. 7, 1899.

[2] Boston M. and S. Jour., Mar. 16, 1900.

[3] Lancet, Sept. 16, 1899.

L. Ferrannini [1] reports 4 cases of anomalies in the form of the body, associated with **cardioptosis.** It has been commonly taught that displacement of the heart downward is the result of mechanical influences which force it downward, examples of such causes being tumors, pleural effusions, etc. He, however, agrees with Rummo that there is a primary essential ptosis of the heart. In the 4 cases reported the cardiac dullness was of normal extent, but both the upper and lower borders were lower than normal, and there was decided epigastric pulsation. In all there was mitral stenosis. The patients showed abnormalities of growth and other signs of imperfect development of the skeleton and large vessels, and Ferrannini believes that the cardioptosis was caused by congenital imperfections in the vascular system, and by the mitral stenosis, which he considers to have been congenital. The loss of elasticity in the vessel walls and the enlargement of the heart allowed of the dropping of the heart downward.

**Pericarditis.**—O. Damsch [2] has investigated the **position assumed by pericardial exudates** by injecting solutions of agar into the pericardium after removing the fifth and sixth costal cartilages and introducing a trocar into the pericardium, the subject being held in the upright posture. He found that small amounts of the solution (about 100 cc.) collected in the lower and anterolateral portions of the pericardium, causing the right anterior portion of the pericardium to become more closely approximated to the chest-wall; with larger amounts of solution the intrapericardial pressure increased and the form of the pericardium became more rounded. The clinical conclusion to be drawn from this is that the first sign of pericardial effusion would be the appearance of an area of dullness in the heart-liver angle, as taught by Rotch. [This is easily explained by a recognition of anatomic conditions. The portion of the pericardial sac over the right auricle is more lax and sac-like than any other part on the anterior surface of the heart.] When the effusion becomes more marked, the dullness increases upward and to the left as well as on the right. The heart, when of normal size, was always found pressed against the posterior portion of the pericardium, the fluid occupying the anterior portion. The heart was not, as is frequently taught, pressed upward and anteriorly, except in one instance, in which the heart was decidedly hypertrophied. In such cases the heart would necessarily be lifted upward and anteriorly, since its increased size causes it to fill the whole space between the anterior and posterior chest-walls; and therefore, since the fluid collects in the lower part, the heart must in such instances be pressed upward, as there is no space for it to go backward. The fact that the fluid collected on the anterior portion of the heart explains the disappearance of the impulse and the weakness of the cardiac sounds in pericardial effusion. From his experiments he decides that the best position for **puncture** is well down toward the lower part of the pericardium. In order to avoid wounding the pleura one should choose a point not at the very depth of the pericardium; hence he decides that puncture in the fifth or sixth intercostal space, next to the sternum, directing the trocar somewhat inward, is the safest method and location of paracentesis. From this point, by moving the trocar partly downward and outward, the pericardium can be entirely emptied. There is no danger of wounding the heart if this point is chosen, since the fluid collects in the lower part of the pericardium and in front of the heart, except when the heart is hypertrophied, and in the latter case the heart is floated upward and out of danger. Undoubtedly in most instances the heart is pushed upward, as almost all cases of pericardial effusion occur in persons who already have some cardiac disease which has led to hypertrophy of the left ventricle.

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[1] Centralbl. f. innere Med., Jan. 6, 1900.

[2] Zeit. f. klin. Med., Bd. XXXVIII, Hefte 4, 5, u. 6.

E. Eisenmenger[1] believes that there is no definite entity which corresponds to Pick's **pericarditic pseudocirrhosis** of the liver. He believes that there is no proof that circulatory disturbance of the liver produces fibroid overgrowth which leads to ascites by interference with the portal circulation. He contends that the hyperplasia that is found in congestion affects the central vein only, and can not produce ascites. The ascites occurring in pericarditis, which suggests cirrhosis of the liver, is due, he believes, to torsion, compression, or angulation of the inferior cava by pleural exudate, to adhesions of the pericardium in the mediastinum, or to a localized peritonitis about the fissure of the liver. [We have seen ascites in cases of cirrhosis secondary to cardiac disease and independent of pleural effusion or any other cause than the hepatic disease and the cardiac weakness.]

**Chronic Endocarditis.**—R. C. Cabot[2] reports his **clinical study of 186 cases** of valvular disease of the heart. He describes the method of recording the clinical symptoms used by him. He emphasizes the fact that aortic regurgitant murmurs were commonly heard most clearly to the left of the sternum. Of 44 cases, the murmur was heard best to the left in 36, and was loudest on the right in only 4. He notes the difficulty in diagnosing aortic stenosis, and doubts whether it is ever proper to diagnose mitral stenosis when there is definite evidence that aortic regurgitation is present, because of the similarity between the Flint murmur and the murmur of mitral stenosis. He considers that the apex-beat is not represented by the point of strongest impulse of the heart, but is usually the point furthest downward to the left in which pulsation can be felt. [Though there is undoubtedly much difficulty in establishing the existence of mitral stenosis in a case in which there is aortic regurgitation, we can not agree with the author's extreme point of view. Often the recognition of the combined valvular condition is easy.]

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[1] Wien. klin. Woch., Mar. 15, 1900.

[2] Boston M. and S. Jour., Feb. 1, 1900.

**Mitral Stenosis.**—Huchard,[1] in discussing the etiology of mitral stenosis, states that he considers **pure stenosis** to be **congenital** and frequently due to hereditary syphilis. In the case reported in this paper there was a distinct history of maternal syphilis. Huchard has observed the same association repeatedly, and has often noticed other malformations in these cases. He insists that mitral stenosis is frequently overlooked because of the common absence of auscultatory evidences of the disease. In cases of hemiplegia, particularly in young women, an origin in mitral stenosis and consequent embolism should always be thought of and looked for.

L. L. Ferannini [2] discusses the **three-toned rhythm** of mitral stenosis—that in which a second diastolic tone is heard. In the 9 cases in which Ferannini has observed this, mitral stenosis was always present in some degree. In order to test the origin of the third sound he gave various drugs and used mechanical measures. Muscular movements caused the disappearance of the third sound and the appearance of a loud systolic murmur; this sound also disappeared after compression of the crural arteries. Distention of the stomach caused it to increase somewhat. As to the effect of drugs, he says that alcohol, strophanthus, and digitalis caused the tone to disappear, and the presystolic murmur to increase in intensity, together with increase in blood pressure. Atropin and amyl nitrite caused the sound to disappear; hence its increase seemed to be dependent upon decreased blood pressure, its disappearance upon increased blood pressure. His explanation is that when the blood pressure is increased, the mitral valves are opened more violently and pressed out of the way of the blood current and against the walls of the ventricle, hence they have no opportunity to vibrate; when the blood pressure is decreased, the mitral valves project into the blood current, and hence produce vibration. This three-toned rhythm is believed to be characteristic of mitral stenosis, but indicates a mild degree of stenosis; if the opening is contracted, the leaflets become so much deformed or so adherent that they can not be thrown into vibration.

T. H. Green [3] describes the case of a boy of 19 who was first seen because of **repeated and severe hematemesis.** The vomiting of blood had begun 2 months before, when he suddenly brought up about a quart of clotted blood. The following evening a smaller quantity was vomited, and about a month later blood was again vomited. He had no epigastric pain, but had swelling of the legs and abdomen and enlargement of the spleen. He was profoundly anemic, but improved at first on treatment. Later he had severe hematemesis and passed blood from the bowel, and the hematemesis continued daily for 5 days, when the boy died. At necropsy the heart was found enlarged and the mitral orifice much stenosed with recent vegetations; there were also some vegetations on the aortic and tricuspid valves and the heart was fatty. There was **thrombosis of the splenic vein** throughout the whole course, from the spleen to the junction with the superior mesenteric vein.

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[1] Jour. de méd., Feb. 10, 1900.

[2] Centralbl. f. innere Med., Aug. 10, 1899.

[3] Brit. Med. Jour., Nov. 18, 1899.

F. P. Henry [1] describes a case of **mitral stenosis with relapsing fever** of nonmalarial origin. The patient was a girl of 19, who had a typical mitral stenosis, and whose fever assumed a distinctly intermittent type, with paroxysms occurring about every 6 days. She had no evidences of malaria. The cause of the fever and of the peculiar intermittency could not be determined. [A somewhat similar case occurred in our experience. In this instance there was extreme congestion of the left lung, and the fever was thought by us to be due to the pulmonary disease—a low grade of bronchitis or catarrhal pneumonia.]

**Mitral and Tricuspid Stenosis.**—T. L. Charbourne [2] describes a case of combined tricuspid and mitral stenosis with adherent pericardium in an epileptic woman who had been 5 years under observation and had had occasional slight attacks of incompensation. In her final illness she had great loss of compensation. The heart signs were those of double mitral disease; the tricuspid stenosis was not suspected during life. The postmortem, however, showed both mitral and tricuspid orifices so narrow as to admit only the tip of the index-finger, and the edges were thickened and almost cartilaginous.

**Pulmonary Stenosis.**—A. Kasem-Beck [3] describes a case of **acquired stenosis** of the pulmonary artery. This occurred in a man of 50, who presented marked signs of cardiac insufficiency, with enlargement of the heart to the right and above; a systolic thrill over the body of the heart, most pronounced in the second left intercostal space; and a very loud systolic murmur, heard all over the heart, but also most pronounced in the second left intercostal space. The diagnosis during life was mitral stenosis. Pulmonary stenosis was thought of, but owing to its rarity, and to the fact that mitral regurgitation not infrequently produces the signs mentioned, the most satisfactory diagnosis was considered to be insufficiency of the mitral. The postmortem showed nodules in the pulmonary artery, scars in the wall near the pulmonary valves, and adherence of the pulmonary valves to this fibrous tissue. There was a relative insufficiency of the mitral valves, but no other organic changes. The tricuspid was thickened. The disease of the pulmonary artery was **syphilitic.**

**Treatment of Chronic Endocarditis.**—Sansom [4] considers that **digitalis in** cases of **mitral stenosis** often does harm by increasing the ventricular action; this is not necessary on the left side, and the result is an overtaxing of the right ventricle. In these cases bleeding is often valuable, and after relief of tension in this way, digitalis may in some cases do good; but he considers caffein or convallaria better, the latter especially. If the heart action is slow, belladonna may be beneficial. Massage and exercise, with systematic bathing, are valuable. [Our own experience corresponds very closely with that of Sansom. We have found strophanthus with bromids a useful combination in such cases of mitral stenosis in which digitalis apparently aggravates the symptoms.]

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[1] Am. Jour. Med. Sci., July, 1899.

[2] Am. Jour. Med. Sci., Mar., 1900.

[3] Centralbl. f. innere Med., June 9, 1900.

[4] Clin. Jour., Jan. 21, 1900.

H. Bosse [1] reports the results of his use of the **dialysate of digitalis** in cases of myocarditis, valvular heart disease, pulmonary emphysema, and interstitial nephritis. This preparation is absorbed more readily than others, and to this he attributes the lack of cumulative effects. Even nausea was noticed in but one case, and then it was slight; and the effect upon the dyspnea, the cardiac action, and dropsies was excellent. The most notable thing was the rapid increase of the urine in a number of cases from about 200 cc. a day up to several thousand. In one case the patient passed, after about 10 days' use of the drug, as much as 6000 cc. of urine a day. Bosse considers it too early to express any positive opinion concerning this preparation, but believes it will prove valuable. The dialysate is made from freshly gathered plants.

Brieger [2] has studied the chemical and physiologic characteristics ot the **arrow poison of the Wakambas.** Its physiologic action is entirely similar to that of digitalis; it seems to be chemically of the same constitution as the arrow poison of the Somalis, and the physiologic effects are the same. Contrary to the teaching of some authors, Brieger found that the bark of the dawa is not an antidote to the Wakamba poison. He recommends therapeutic study of the Wakamba poison, as it may prove a valuable substitute for digitalis.

A. Morrison [3] recommends the use of small doses of **mercury** in cardiac disease. He believes that the remedy acts by relaxing the vasomotors, and is thus valuable in conjunction with direct cardiac stimulants, such as strychnin and digitalis. He gives doses of $\frac{1}{5}$ of a grain or more every 2 or 4 hours for 4 or 5 days, alone at first, and afterward combined with digitalis. Ptyalism rarely occurs. Diuresis occurs after 4 or 5 days. It is more useful in mitral than in aortic disease, and is of little value when there is actual nephritis. Diuresis is largely due to the direct action on the kidneys.

A. Meyer [4] discusses the use of the **Nauheim baths** at home with artificially prepared salts. In order to demonstrate the effect of these baths he investigated their influence upon his own person, and found that the pulse was reduced from 76 to 62; there were increased respiration and a sense of exaltation; the cardiac and liver dullnesses decreased, the cardiac dullness to the extent of about $\frac{1}{2}$ of an inch. [This certainly suggests to one that the result was due to increased respiration, since the heart was normal.]

H. Hensen [5] discusses the previous work that has been done concerning the effects of **carbonic acid baths** upon the circulation, and in especial upon blood pressure, and decides that the use of unsatisfactory instruments is responsible for the variations in the results. He made

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[1] Centralbl. f. innere Med., July 8, 1899.

[2] Deut. med. Woch., Sept. 28, 1899.

[3] Lancet, Oct. 28, 1899.

[4] Med. Rec., May 26, 1900.

[5] Deut. med. Woch., Aug. 31, 1899.

64 determinations of blood pressure with Riva-Rocci's instruments which he considers wholly satisfactory. The cases investigated were instances of valvular disease of the heart, myocarditis, and weak heart. He decides that the blood pressure is almost always increased. The frequency of the pulse varies, as does the effect upon the cardiac dullness. The urine he found practically constantly increased, the increase being especially notable when the 3-hour period which included the bath was compared with the previous 3-hour period. The increase in blood pressure is attributed to increased cardiac work, and is, of course, a warning that the baths must be used with the utmost care in cases of disease of the vessels in which there is tendency to hemorrhage.

W. Ewart [1] has investigated the effects of **inhalation of carbonic acid gas** in cardiac disease. It should be free from all traces of CO, and air should be simultaneously administered. It is inhaled by holding the mouth-piece between the teeth, the lips being kept open to admit air. The effects produced are some feeling of internal warmth and desire to breathe, particularly to exhale, some excitation of the circulation and kidneys, and at times headache. Ewart has found that there are often a rapid decrease or an entire cessation of cardiac distress or pain, a feeling of increased freedom of respiration, an increase in the depth of breathing, an improvement of the pulse, and an apparent improvement in the general expression and in the complexion. Repetitions of the inhalations produce progressive improvement in the patients' condition. It may be tentatively tried in any form of cardiac disease with varying results, but is particularly valuable in mitral cases and in aortic stenosis. It may be safely used in angina pectoris. [Carbonic acid gas is a powerful vasomotor stimulant; its use in certain kinds of cardiac disease may therefore be harmful. This note of caution we believe to be necessary.]

F. G. D. Kerr,[2] in discussing the **hot-air treatment,** mentions valuable results in gout, rheumatism, sciatica, and other conditions, in which the treatment is frequently used, and also recommends it in chronic phlebitis. He states that he has used it in heart disease when the patients had cold hands and feet, but that it is often inadvisable to use so powerful a treatment in cardiac cases. Sometimes it has proved valuable in cardiac debility and anemia.

Desplats [3] reported to the Medical Society of Lille a case in which, with severe cardiac insufficiency, there was great enlargement of the abdomen, apparently from ascites. An attempt at tapping in the usual place was without result. He tapped higher, and **removed about 1200 cc.** of a red liquid, which coagulated *en masse.* It seemed to consist of **pure blood,** and he concluded that he had tapped a much enlarged spleen. He describes the result of the tapping as "veritable resurrection." He describes another case in which he had accidentally tapped a greatly enlarged spleen. Angier, in discussion, reported a case in which he had accidentally tapped the right auricle, with great improvement in the symptoms. Franchomme had also accidentally tapped what was probably the spleen, withdrawing between 1 liter and 1½ liters of blood. [All these cases illustrate the occasional fortunate result of grave errors. The many unfortunate results of similar errors must not be forgotten.]

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[1] Brit. Med. Jour., Oct. 28, 1899.

[2] Practitioner, Oct., 1899.

[3] Gaz. hebdom. de méd. et de chir., Feb. 8, 1900.

Abée [1] has previously advised the use of a **plate over the precordia** for the purpose of giving support to an enlarged heart. Marked cardiac hypertrophy is likely to drag the heart downward and to interfere with its action. Abée finds that the application of the plate in such cases causes the outlines of the heart to become smaller, and induces an inward retraction of the apex-beat, a slower and increased fullness of the pulse, and apparently better action of the lungs. He recommends the treatment of attacks of **cardiac asthma** by applying a disc-shaped compress over the precordia. The effect is stated to be very good. The idea was suggested by observing that patients with cardiac asthma often relieve the attack by pressing their hands strongly against the precordia.

**Alterations in Rhythm.**—P. Zenner [2] treated a case of severe **tachycardia,** the cause of which was unknown, by hypodermic administration of apomorphin. The attacks were controlled for a long time, but finally the drug lost its effect. The patient ultimately died from hemiplegia.

R. F. Chase [3] describes a case of **bradycardia** in a man of 75 who had no previous history that seemed important except that he had used tea, coffee, and tobacco to excess. He had some cyanosis and edema of the lungs. There was no evidence of involvement of any organ except the heart. This organ showed no enlargement, but the sounds were labored and there was a systolic murmur at the apex; the pulse was from 26 to 34. There was no atheroma. The man died 2 months after he was first seen, having syncopal attacks in the latter part of life, with cyanosis, and, toward death, some fever. Chase reports a series of cases of bradycardia which he has collected from the literature. He considers a pulse-rate under 60, if synchronous with the heart contractions, to constitute bradycardia. Cases of the idiopathic type, to which this one belongs, usually result in death.

A. H. Whitridge [4] reports a case of **bradycardia with intermittent albuminuria,** the pulse sinking as low as 32. The man had a rheumatic and alcoholic history, but he had had a lipoma removed from his neck some time before, and the possibility of inclusion of the pneumogastric nerve in the scar of the wound is considered by the author as possible explanation of the case. Arteriosclerosis of the coronaries was very likely present, however, and may have caused the symptom.

Ebstein,[5] in discussing **arhythmia** and diseases of the myocardium without arhythmia, concludes that irregularity of the pulse indicates increased demand upon the heart when its cavities are overfilled and there is disturbance of the relation usually existing between the general resistance of the circulation and the power of the heart muscle. Arhythmia may be found in various forms of cardiac diseases, but is most common in cases of myocardial disease.

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[1] Deut. med. Woch., Jan. 25, 1900.
[2] N. Y. Med. Jour., Mar. 3, 1900.
[3] Boston M. and S. Jour., Feb. 8, 1900.
[4] Boston M. and S. Jour., Mar. 29, 1900.
[5] Deut. Arch. f. klin. Med., Sept. 29, 1899.

**Diseases of the Myocardium.**—A. Bier,[1] in considering the cause of the **hypertrophy of the heart** and vascular changes **in nephritis,** states that he believes these changes to be compensatory, and that they occur largely because it is difficult for the kidneys to secrete proper amounts of urine when diseased, and the blood pressure is increased by cardiac hypertrophy for the purpose of adding to the filtration through the kidneys. The actual cause of the hypertrophy is probably some chemical substance, though Bier suggests that the internal secretion of the kidneys may have some effect.

Rénaut[2] discusses some experimental work which he has carried on in animals concerning the **effect** upon the myocardium **of inoculation with toxins.** The experimental myocarditis resulting from diphtheria toxin is practically the same as that seen in the human subject. A much less severe grade is caused by the typhoid toxin, and that produced by pneumonia resembles the typhoid form. Myocardial changes in these conditions may be due either to the toxins or to the organisms themselves, as the typhoid bacillus and Diplococcus pneumoniæ have been seen in the lesions. The clinical course of the disease is similar in the three conditions—a preliminary period of cardiac excitement, followed by a period of weakness of the heart and low pulse-tension. The myocardial changes that affect the parenchyma have no definite clinical results, while the interstitial changes always cause decided and definite symptoms, consisting of prostration, gastro-intestinal disturbance, and rapid and irregular heart action. Suppurative myocarditis clinically resembles ulcerative endocarditis, but there may be intense pain in the precordia. It is questionable whether the patient may recover completely from an attack of acute myocarditis. Remnants of the former trouble could be seen in animals as long as 2 years after its experimental production. [It has been our opinion that slight remnants of this sort may be the cause of cardiac weakness, perhaps years after the original disease, and with no intervening evidences of cardiac disease.]

F. J. Poynton[3] describes a case of **diphtheria,** one of **rheumatism,** and one of **chorea** in which there was decided **myocarditis,** which apparently was primary and not the result of endocarditis or pericarditis. He insists that such cases demonstrate the importance of giving due attention to all cardiac symptoms in acute disease, as they show that even when signs of valvular lesions are absent there may be severe cardiac involvement. This is particularly true in children with rheumatism, and it must be remembered that in adults with rheumatism it is not necessary to have signs of valvulitis in order to demonstrate the existence of cardiac involvement.

E. A. Darling[4] discusses the **effects of training** upon the Harvard University crews. The effects upon weight were usually more or less marked reduction; particularly after time rows there was usually a loss of weight, which was soon afterward regained. The temperature was usually variable, but he was not able to observe the remarkable drops in temperature seen by Williams and Arnold in the Marathon runners. In most of the men the examination of the heart showed prominence of the precordia, and, in the earlier stages of the training, progressive enlargement affecting both sides of the heart; later there was considerable shrinkage, which on the left side reached about the normal, the right side being reduced somewhat less than the left. The changes in the size of the heart were probably due both to hypertrophy and dilation. The heart-sounds showed 3 groups of changes. In one no marked abnormalities were found; in another group blowing murmurs developed after time rows and after the race; in the third the heart showed abnormalities both before and after unusual effort. In one case there was decided improvement during the course of the training—irregularity, accentuation of the second pulmonary sound, and embryonic rhythm disappearing. This man probably had a cardiac lesion, as he previously had had rheumatism and typhoid fever. The men who showed abnormalities in their hearts before effort finished the race in as good condition as the others. The pulse was always of high tension after effort. As to the kidneys, traces of albumin were found in a large proportion of the men under ordinary conditions, 48 of 83 specimens showing albumin in the 24-hour specimens, and usually casts and epithelium were present also. After time rows and races the specimens invariably showed albumin and hyaline and granular casts, sometimes also renal cells and red blood-corpuscles. Darling insists that the results of examination of the heart showed that the heart must be looked upon as a muscular organ, and that in violent and prolonged exercise it shows the same changes as do other muscles. As to overtraining, he suggests that while the true condition at the bottom of this is obscure, one factor may be excessive demand upon the heart; another factor is probably the effect upon nutrition which results from an improper diet or from disturbed digestion. A third factor may be simply overwork, and the fourth factor is undoubtedly a nervous one. This, while intangible, is probably important, nervous excitement and the like undoubtedly having a good deal of influence.

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[1] Münch. med. Woch., April 17, 1900.

[2] Presse méd., July 29, 1899.

[3] Lancet, May 12, 1900.

[4] Boston M. and S. Jour., Aug. 31 and Sept. 7, 1899.

A. Stengel[1] discusses the **effect of athletics** on the heart and circulation. He presents a series of charts illustrating the condition of the heart before and after athletic exercises in men training for various contests. The untrained athletes usually show some cardiac dilation and often murmurs, which are probably the result of acute dilation of the conus arteriosus after severe exertion. These were not often observed in the trained athletes. In some cases marked hypertrophy was observed as a more remote effect of athletics, and Stengel considers that a number of cases which he has seen in middle-aged men were a result of changes in the heart muscle which resulted from overexertion in athletics in early life. Stengel considers that athletics raise the blood pressure through the cardiac hypertrophy, if for no other reason. Overdistention, hypertrophy, and overaction of the heart he finds common after athletic contests, and as a result of continued athletic strain in those who are not thoroughly seasoned to the work; and he thinks that there should be careful medical supervision of men during the athletic period of life, and that the exercises should be gradually and not suddenly discontinued.

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[1] Am. Jour. Med. Sci., Nov., 1899.

I. B. Yeo[1] discusses **cardiac strain in adolescence** and in middle age. He describes first a form which occurs in girls at about puberty, due to rapid growth and educational strains, the muscular force of the heart not developing with a rapidity equal to the growth of the rest of the body. It is characterized by palpitation, tendency to dyspnea, fainting and giddiness, with some edema of the ankles, pain in the chest, and cardiac murmurs. The treatment should be rest, proper food, and cardiac and blood tonics. In young men the causes are chiefly excesses in physical exercise, in tobacco, and in sexual indulgence. The treatment is chiefly rest, tonics, and removal of the cause. The chief causes in middle age are attempts to perform the same amount of work or of physical exercise that was indulged in in earlier years.

**Treatment.**—H. B. Allyn[2] discusses the value of **blood-letting** in the treatment of dilation of the heart. It is particularly valuable when the right heart is much dilated and there is considerable arterial tension. It lessens the volume of blood, and it also removes poisonous products of metabolism. Venesection should usually be done earlier than it is ordinarily undertaken, and more blood should be withdrawn than is common. It should not be used in very young or very old subjects. If it is feared that the shock may be too great, leeches may be used on the epigastrium. The amount withdrawn in venesection should be from 6 ounces up to as much as 18 ounces.

Huchard,[3] in discussing the **treatment of cardiac sclerosis,** divides the disease into the stages of excessive arterial tension, or the presclerotic stage; the stage in which the blood-vessels become involved greatly; and the final stage, in which the blood-vessels of the heart become markedly implicated and the heart itself fails. The treatment in the first stage should consist largely in the avoidance of stimulating beverages, including tea and coffee, and of liquids, except diuretic mineral water, and the use of a milk diet with small doses of lycetol or piperazin. Digitalis and all drugs increasing the arterial pressure should be excluded. Diuretics, however, such as caffein, are advisable, and mild purgatives are useful. Also, all drugs that tend to decrease elimination by the kidneys should be excluded, patients should not be allowed to dwell in high altitudes, and the use of tobacco should be prohibited. In the second stage the same dietetic and hygienic measures should be continued. The iodids become the most important drugs unless heart failure is evident, in which case digitalis or spartein should be used. He considered it very important, in order to increase the elimination of toxic materials and to overcome insomnia, to use a rich milk diet in this stage. If edema of the lungs comes on, venesection should be performed.

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[1] Edinb. Med. Jour., July, 1899.
[2] Univ. Med. Mag., Dec., 1899.
[3] Jour. de Praticiens, Dec. 23, 1899.

Huchard,[1] in discussing the use of **digitalis and caffein** in cardiac disease, recommends digitalis in extreme dilation, and considers that it is not contraindicated except in advanced stages of cardiac disease. In such cases caffein usually does better. The latter is also more effective in acute inflammations with severe cardiac weakness, and in acute Bright's disease. Often the preliminary administration of caffein, with subsequent administrations of digitalis, will produce the best results.

W. H. Thomson,[2] in discussing the therapeutics of heart disease, states that his treatment for **acute inflammatory conditions** is rest, leeches, and **sedative applications** over the precordia. He gives aconite and usually excludes digitalis. In acute degenerations of the heart digitalis should be absolutely excluded, as it is likely to do harm, and one should give strychnin, caffein, or camphor injections. The sequelæ of acute endocarditis should be guarded against by prolonged rest and continued administration of aconite. The pain and dyspnea which follow pericarditis are often greatly relieved by strapping the chest. Belladonna is also useful. Thomson insists upon the **importance of myocardial degeneration** in causing the cardiac troubles of those beyond middle life. In the treatment it is essential to keep up a good flow of urine, and to overcome any disturbances of the gastrointestinal tract. The former purpose is answered by using Kemp's irrigator with salt solution at a temperature of 110° F. In the stage of marked failure of compensation digitalis with nitroglycerin should be used.

**Rupture of the Heart.**—A. W. Hoisholt[3] reports 2 cases of **spontaneous rupture** of the heart **in insane subjects.** In one case the patient was found dead in bed without any signs of agonal disturbance, and no sounds had been heard by others in the dormitory. In the other case the symptoms came on 6 hours before death. They were chiefly severe pain, with dyspnea and cyanosis. In this case there was degeneration of the heart muscle and of the vessels of the heart. A. Sutcliffe[4] reports a case of rupture of the heart which occurred in an insane man 53 years of age, who had been in apparently good condition. He was found dead in the laboratory. The pericardium was found filled with blood, the heart was pale and fatty, and there was a rupture in the left ventricle. There was no evidence of syphilis or of fibroid change. A. Kalenberg[5] describes the case of a woman who was carrying a heavy weight and lost her balance, and apparently overstrained herself in attempting to regain her equilibrium. She fell, and was found speechless and very dyspneic, dying almost at once. She had apparently been healthy before. The necropsy showed a large amount of blood in the pericardium, and in the left ventricle near the junction with the auricle a rupture, which was about an inch long externally. The heart was fatty, but the valves were unaffected, and the large blood-vessels showed no decided atheroma.

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[1] Méd. moderne, Feb. 17, 1900.
[2] Med. Rec., Mar. 17, 1900.
[3] Occidental Med. Times, Oct., 1899.
[4] Brit. Med. Jour., Jan. 20, 1900.
[5] Brit. Med. Jour., Dec. 23, 1899.

**Syphilis of the Heart.**—P. M. May [1] describes a case in which sudden death occurred, and in which the postmortem examination showed a large yellowish-white mass between the two ventricles about the size of a pigeon's egg, and a smaller one in the pulmonary valves. There were numerous scars in the liver, and microscopic examination of the mass in the heart showed that it was a gumma.

## DISEASES OF THE ARTERIES.

T. Clifford Allbutt [2] discusses **senile arterial plethora,** a condition which he describes as occurring in elderly persons, and as having somewhat characteristic symptoms, the principal objective symptom being increased arterial pressure, while the patient complains of nervous symptoms, which are chiefly vertigo, panting, depression of spirits, disturbance of sleep, and irritability and capriciousness of temper. Certain seizures may also occur which simulate to a greater or less degree epilepsy, apoplexy, and syncope. The symptoms are most marked in the morning and decrease toward evening. The condition is curable by proper treatment in the earlier stages. Restless apprehension and fretfulness are common complaints, and so-called senile hysteria, which occurs in persons who are not previously neurotic, is frequently due to this condition. Allbutt considers that the blood is probably primarily at fault, and that the alteration may produce either arteriolar contraction or increase of density, which causes consequent increase of friction. The duration of systole is not necessarily prolonged by the increase of pressure, but ultimately, if the vessels are not normal, organic changes will appear in the heart. In some of these cases there is no evidence of marked arteriosclerosis, and atheroma may be entirely absent. Allbutt does not consider that the increase of pressure is due to changes in the arterial walls.

**Arteriosclerosis.**—G. Capsammer [3] discusses **Gaertner's tonometer.** The essential of the instrument is that the blood is pressed out of the finger, and the pressure caused by the return of the blood is measured. Capsammer finds that the blood pressure varies widely in ordinary conditions; it sinks during anesthesia, and after copious hemorrhages, removal of exudates, the opening of the abdominal cavity at operation, etc. In one case traction upon the sciatic nerve during operation caused a marked fall, while in other cases traction upon the second division of the fifth nerve and upon the brachial plexus caused increase in pressure, probably through reflex stimulation. The normal pressure seems to be between 100 mm. and 130 mm. The lowest pressure compatible with life was about 60 mm.

H. Weiss,[4] in discussing the use of Gaertner's tonometer in the determination of **alterations in the blood pressure,** states that increased tension is found in arteriosclerosis, even when there is no determinable alteration in the peripheral vessels. In a man with slight signs of arteriosclerosis the reading of the tonometer was as high as 240 mm. of mercury. This man died of apoplexy after emotional excitement. If arteriosclerosis is present and the tonometer reading is low, it indicates failure of heart power. The effects of cardiac stimulants may be seen after using this instrument. In nephritis the reading is usually high.

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[1] Brit. Med. Jour., Dec. 23, 1899.

[2] Phila. Med. Jour., April 14 and 21, 1900.

[3] Wien. klin. Woch., Dec. 21, 1899.

[4] Münch. med. Woch., Jan. 16 and 23, 1900.

J. R. Ambler [1] investigated 100 bodies postmortem in order to determine the frequency of **atheroma of the aorta.** Atheroma was found in 69 % ; in mild degree in 10 males and 16 females, more marked in 17 males and 9 females, and very strongly marked in 14 males and 3 females.

M. Lapinsky [2] describes 2 cases of **trophic alteration** in the vessels **in the course of peripheral neuritis.** The first patient had peripheral neuritis of all 4 extremities. The changes in the vessels were indicated by cyanosis, dilation and tortuosity of the vessels, and the occurrence of spontaneous subcutaneous hemorrhages. The vessels also felt thick and hard, and in a piece of skin which was removed there was seen a marked thickening of the walls from endarteritis. There were no local causes of disease of the vessels other than the changes in the nerves, and the vessel changes were localized to the areas supplied by the diseased nerves. Also, the changes in the vessels were coincident with those in the nerves, and the involvement of the vessels was seen in widely separated areas, which could not be influenced by any local cause, such as direct injury. Lapinsky decides for these reasons that the changes were due to alterations in the trophic nerves supplying the vessels.

**Treatment.**—Cantru [3] describes the effects of **abdominal massage** as lowering of arterial pressure and reduction in the frequency of the pulse, followed by free diuresis. These produce general improvement in the circulation. He believes that massage is indicated in arteriosclerosis and in conditions leading thereto. In any other conditions producing vasoconstriction it may act as a cure, and also may prevent the occurrence of arteriosclerosis when conditions likely to lead to it are present.

H. Beates [4] insists that the belief that circulatory stimulants are contraindicated in the degenerative vascular lesions of the aged because of the supposed increase in the arterial tension is erroneous. He insists that when the propelling function of the arteries is lessened, the tension can not be increased ; therefore he recommends the use of **digitalin,** which should be used only when made by a reliable firm. He finds this very valuable in the circulatory failure of old age.

A. Morrison [5] believes that **mercury** may with advantage be much more freely used in the treatment of cardiac failure due to arteriosclerosis. He reports a series of cases which improved greatly after using the old digitalis, squills, and blue pill combination.

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[1] Edinb. Med. Jour., Dec., 1899.

[2] Zeit. f. klin. Med., Bd. XXXVIII, Hefte 1, 2, u. 3.

[3] Jour. de Praticiens, Aug. 12, 1899.

[4] Phila. Med. Jour., Nov., 1899.

[5] Lancet, June 30, 1900.

**Aortitis.**—[French authorities have always regarded aortitis as a much more frequent affection than has been admitted by others. Undoubtedly many of the supposed instances were cases of atheroma. Those that have been diagnosticated but not demonstrated postmortem are of doubtful character, and, taking all the evidence into consideration, there is little proof that the disease is frequent or clinically recognizable.] M. Potain [1] describes a case of **malaria** in which there was present what he calls an **acute transitory aortitis.** The diagnosis of the latter condition was based upon the fact that there was marked increase of the dullness due to the aorta, more especially to the left of the sternum, without any coincident marked increase of the cardiac dullness. The aortic dullness afterward decreased, and the condition became normal. Potain considers that the increase in the dullness was due to dilation of the aorta and a stretching of this vessel, a result of the weakness produced by the malarial intoxication. He thinks, however, that so much change in this vessel could not have occurred without some organic alterations in its walls. He therefore considers the case one of aortitis.

Lancereaux [2] describes a case of malarial aortitis. He believes that malaria is a frequent cause of disseminated aortitis. Of 37 persons having aortitis of this form who had come under Lancereaux's observation, 26 had a history of intermittent fever, and 8 had lived in malarial districts. The lesions are seen chiefly in the descending portion of the arch, and begin in the adventitia. Later the media atrophies, the intima becomes hypertrophied, and plates form. Angina pectoris may result, as may also aneurysm. Of the patients whom Lancereaux has observed, 8 had aneurysm, and all had anginoid attacks. Laveran severely criticized the report, and expressed serious doubt of the occurrence of malarial affections of the aorta that can be diagnosed clinically or that have any distinctive pathologic features. [Authorities in this country and elsewhere, except in France, agree with Laveran.]

**Rupture of the Aorta.**—Hussenet [3] describes a case of spontaneous rupture of the aorta, the man dying suddenly. Autopsy showed that the pericardium was distended with a clot, which was shown to be the result of rupture of the aorta. This vessel presented ulcerations on the deeper surface, but there was no aneurysm. One of the ulcerations near the valve showed a large perforation.

**Aneurysm.**—H. A. Hare and C. A. Holder [4] present a **statistical study** of aneurysm of the aorta, particularly directing their attention to the determination of the most frequent situation, the commonest cause, the direction of development, its frequency in the two sexes, and the most common symptoms and causes of death. Of 570 cases of aneurysm of the ascending arch, 504 were sacculated. Four hundred and sixty-six occurred in men; only 78 in women. Aneurysm is most common between the thirty-fifth and the forty-fifth years of life. The important etiologic factors were syphilis, alcoholism, rheumatism, and trauma. Death occurred from a rupture of the sac, or as a result of pressure on important organs. Most of the sacculated aneurysms ruptured into the pericardium, though a number perforated into the pulmonary artery; others ruptured into the right auricle, the left auricle, the right or left ventricle, the superior vena cava, the pleural cavity, the lungs, the posterior mediastinum, or externally. In cases in which a note was made of its condition the heart was said to be normal in 58 and hypertrophied in 29. They collected 88 cases of aneurysm of the transverse portion of the arch and 110 of the descending portion. There were also 169 in which the location was not well described. Aneurysm is unquestionably most frequent in the ascending portion of the arch. The transverse and descending portions of the arch are about equally affected. They believe that death does not so frequently occur from rupture as from the results of pressure, and that syphilis is not so important a factor as it is often considered.

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[1] Bull. Acad. de méd., No. 30, 1899.

[2] Semaine méd., July 5, 1899.

[3] Arch. de Méd. et de Phar. Milit., Oct., 1899.

[4] Am. Jour. Med. Sci., Oct., 1899.

A. Heller [1] believes that aortic aneurysm is frequently of **syphilitic origin,** and that aneurysms resulting from this cause are much more frequent than those due to other conditions. He has found 85 % of cases of aneurysm to be syphilitic. The lesions produced by syphilis are marked cellular hyperplasia and the presence of giant cells, the growth being chiefly about the vasa vasorum. Later fibrous tissue forms, and causes obliteration and necrosis of the vasa vasorum. The gross appearance of syphilis of the aorta is distinguished by the presence of small areas with pit-like central depressions and wrinkled borders, with thickening of the intima.

W. M. L. Coplin and E. A. Thornton [2] report a case of aneurysm of the aorta in which there was apparently an **old rupture into the pulmonary artery** and a recent fatal rupture into the pericardium. The patient had presented no signs that seemed to bear any definite relation to the rupture into the pulmonary artery, but some weeks before death he had had an attack of syncope, from which he recovered slowly, and it was possible that the rupture occurred at that time.

W. Porter [3] discusses the early **diagnosis of aneurysm of the aorta,** directing attention to the laryngeal changes, and particularly to the hoarseness and monotonous tone of the voice and the inability to produce high notes. Dyspnea is a very frequent symptom, and may be laryngeal, resulting from paralysis of the cords. Pain in the neighborhood of the fifth and sixth dorsal vertebræ is a common sign, and auscultation along the left scapula may show a diastolic murmur in the arteries not heard elsewhere, or one may observe a systolic murmur, caused by the throb of the sac against the left bronchus; and auscultation of the brachial artery may disclose a systolic thumping sound similar to that heard in aortic insufficiency. Porter recommends examination for aortic aneurysm by introducing a large bougie into the esophagus, the lower end being very distensible or being covered with gold-beaters' skin. The lower end is passed down to the site of the aneurysm, the impulse of the aneurysm being conveyed to the distended lower end. Also if a U-shaped tube filled with fluid is passed down to the site of the aneurysm, there will be seen a vibration of the fluid, while normally there is scarcely any vibration communicated to it. One may also auscult aneurysms by way of the esophagus, passing into the esophagus a solid sound having a hard disc fastened to the upper end. To this, one may apply the stethoscope; and if a bruit is present, it will usually be conveyed through the solid bougie with great distinctness. [This and other similar methods of direct auscultation through the esophagus have been previously recommended, but it is questionable whether such examinations are sufficiently safe to be practised.]

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[1] Münch. med. Woch., Dec. 12, 1899.

[2] Proc. Path. Soc. of Phila.

[3] N. Y. Med. Jour., Dec. 9, 1899.

M. Auerbach [1] quotes Fraenkel as having said that **tracheal tug and Cardarelli's phenomenon** are found only rarely in any condition other than aortic aneurysm, but that when the tumor presses the aorta firmly against the bronchus, or when the tumor is adherent to both the bronchus or trachea and the aorta, both these phenomena may be present with mediastinal tumor. Auerbach reports 2 cases which exemplify these statements. The first was one of primary carcinoma of the stomach, with metastasis to the mediastinal and other glands. Both Oliver's and Cardarelli's symptoms were present. The postmortem showed no aneurysm, but revealed a carcinomatous infiltration firmly adherent to the left bronchus on the one side and to the aorta on the other. In the second case the diagnosis during life was in doubt. The postmortem showed a mediastinal growth similar to that found in the first case. Nevertheless Auerbach states that tracheal tug must be looked upon as speaking strongly in favor of aortic aneurysm because of the rarity of its occurrence in other conditions.

H. Strauss [2] reported a case of aneurysm of the aorta in which the Oliver and Cardarelli symptoms were present in spite of the fact that the aneurysm was not in contact with the bronchus. It is practically always observed that these symptoms are due to contact with the left bronchus. In this case, however, the mass was in contact with the anterior part of the trachea only.

S. Ritoók [3] describes several interesting observations of the presence of **tracheal tug** and the Cardarelli symptom in cases in which there was not pressure of an aneurysm upon the left bronchus. The most important was an instance of **localized left-sided pleurisy** in the costomediastinal space. The tracheal tug and the Cardarelli phenomenon were marked in this case, but disappeared entirely after aspiration of the fluid. The explanation for their occurrence was that the situation of the fluid was such that it compressed the aorta against the left bronchus, and thus gave rise to the tracheal phenomena. In another case tubercular glands existed about the trachea and bronchi. Some of the glands were adherent on one side with the left bronchus and on the other with the aorta. They had caused marked tracheal tug, and this, together with stenotic breathing, had given rise to a diagnosis of mediastinal tumor which was considered to be possibly an aneurysm. No aneurysm was found, however. In another case there was decided movement of the larynx from below upward and from the right to the left, resulting from pressure and pulsation of an aneurysm of the innominate artery.

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[1] Deut. med. Woch., Feb. 22, 1900.

[2] Berl. klin. Woch., Sept. 18, 1899, p. 843.

[3] Deut. med. Woch., June 28, 1900.

G. Kirchgaesser [1] describes a case in which aortic aneurysm had been diagnosed, and in which the fluoroscope had disclosed the presence of a pulsating tumor near the heart. At autopsy this was found to be due to a **carcinoma of the esophagus,** with adhesions to the aorta. [The difficulty of differentiating aneurysm from other mediastinal tumors can not be too often discussed, and is not wholly overcome by application of the fluoroscope.]

F. L. Classen [2] describes the case of a man of 40 who had spasmodic cough and paralysis of the left vocal cord. The usual physical examination was entirely negative, but examination with the **x-rays showed** what was taken to be an **aneurysm** of the aorta. The examination was made by Williams, of Boston. The postmortem revealed no aneurysm, but disclosed enlargement of the retrotracheal glands. [This is further evidence of what has already been well demonstrated—that the x-rays can not be relied upon exclusively in either medical or surgical diagnosis.]

Huchard [3] reported a case of aneurysm in a man of 58, in which for a long time the only symptom was pain; but because this pain was most marked at a definitely limited point, was increased by change of position, and was not influenced by treatment, and because the man had a history of syphilis, aneurysm of the aorta was suspected, and was finally found. In discussing the **prognosis of aneurysm** Huchard insists that it is of extreme importance to distinguish between those cases of aneurysm with lowered arterial tension and those with increased tension. When the arterial tension is increased, the aneurysm is likely to run a very rapid course. This is particularly the case when there is marked arteriosclerosis or interstitial nephritis. With increased tension regulation of the condition of the gastro-intestinal tract is of great importance, as gastro-intestinal intoxication increases vasoconstriction.

Merklen [4] describes a case of traumatic **neurotic tachycardia** in which aneurysm afterward developed. He believes that the attacks of tachycardia were premonitory of the possible development of aneurysm of the aorta in such cases, the traumatism having caused injury to the nerves about the aorta, and at the same time probably of the aorta itself, and the aneurysm developing as a result of trauma. The tachycardia indicated damage to the nerves about the aorta.

Souques [5] reports a case of aneurysm of the aorta which was interesting because of the existence of a tumor in the upper portion of the left chest, evidently arising from the left side of the horizontal portion of the arch. A point of still greater interest was the occurrence of repeated attacks of **ictus laryngis.**

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[1] Münch. med. Woch., May 8, 1900.

[2] Albany Med. Ann., Oct., 1899.

[3] Gaz. des Hôp., Feb. 16, 1900.

[4] Gaz. des. Hôp., Dec. 17, 1899.

[5] Gaz. des Hôp., Oct. 20, 1899.

M. Weinberger and A. Weiss [1] describe a case of **aneurysm of the descending aorta** in which there was marked pulsation and dullness beneath the manubrium, and on the right a pronounced pulsation in the back. Profuse hemorrhage had occurred repeatedly, and the autopsy showed perforation into the right bronchus. In spite of the large size of the aneurysm, the subjective symptoms had been slight.

L. E. Shaw [2] discusses the case of a man of 26 who was admitted to the hospital in collapse. The history as obtained was entirely negative, and no syphilitic infection could be determined. He died soon after admission, and the pericardium was found filled with blood which came from an aneurysm situated on the first part of the aorta. There was also a gelatinous mass in the apex of the left ventricle about the size of a ten-cent piece, and this seemed microscopically to be **a gumma.**

R. C. Cabot [3] describes a case as one of probable thoracic **aneurysm presenting intermittently** through the sternum. The patient was a woman of 42 who had a history indicating the presence of syphilis. She had had intermittently for 7 years a mass over the front of the sternum. It had the appearance of the stump of a cow's horn, and was about the size of a lemon. It broke spontaneously while under observation and discharged only some purulent material. The mass pulsated with every heart-beat, and the pulsation was slightly expansile. There was a second mass, which was smaller, below the first, at the level of the third and fourth cartilages. There were apparently no other signs of aneurysm, and several physicians who saw her agreed that there was no aneurysm. However, she died with a sudden gush of blood "issuing from her chest." Examination with a probe had shown that there was a small hole in the sternum. X-ray examination had shown that there was a bulging in the region of the ascending arch of the aorta. [The external masses certainly appear to have been gummata, and were so diagnosed; whether an aneurysm was present or not, there is certainly only insecure testimony that it presented intermittently through the sternum.]

E. v. Leyden [4] describes a case of **aneurysm of the abdominal aorta** in which there was an easily palpable round mass which showed expansile pulsation. The diagnosis was perfectly secure. The most interesting fact observed was that there was **diastolic murmur** over the tumor, which was heard as one auscultated upward even as far as the aortic interspace. There were, however, no evidences of aortic regurgitation. The murmur was weaker as auscultation was carried upward, and it was considered a case similar to those previously described in which a diastolic murmur is transmitted from the region of the aneurysm, and is due to the aneurysm. The explanation of the occurrence of the murmur given by Scheele is considered correct: that is, the opening from the aneurysm into the aorta is narrower than the lumen of the aorta above, hence the blood flows during diastole from the aneurysmal opening into a wider space and causes a diastolic murmur.

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[1] Wien. klin. Woch., Feb. 22, 1900.

[2] Lancet, Sept. 20, 1899.

[3] Am. Jour. Med. Sci., April, 1900.

[4] Deut. med. Woch., June 7, 1900.

W. Ewart and J. Jaffrey[1] report a case of uncontrollable vomiting in which a pulsating tumor, associated with a bruit and thrill, was felt in the lower epigastrium. Exploratory operation was done, and a large fusiform aneurysm of the abdominal aorta was discovered which arose just below the diaphragm and extended to about the level of the umbilicus. The stretching of the pyloric end of the stomach over the aneurysm had caused some **obstruction of the pylorus** and interfered with the outflow of the stomach-contents. The stomach was pushed to the right by the operator, and remained in this position without fixation. The woman improved rapidly and the vomiting disappeared, and some time afterward she had become able to take solid food and to move about.

**Treatment.**—T. B. Futcher[2] discusses the literature concerning the treatment of aneurysm of the aorta by **injections of gelatin** solution. He describes 9 cases in which this treatment has been carried out in the Johns Hopkins Hospital. The immediate effect of the injection was frequently severe and protracted pain; sometimes chills occurred from 2 to 4 hours after the injection; and the temperature went as high as 103° F. Suppuration did not occur, and only once was there any local reaction. In no one of the 9 cases treated was there a cure, but Futcher thinks that the treatment is worthy of further trial. In one case of abdominal aneurysm the mass was somewhat reduced in size, and in 7 of the 9 cases the subjective symptoms were considerably improved.

A. Fraenkel[3] describes a case of aneurysm of the aorta in which gelatin injections were used, as perforation was feared. Ten injections were made without causing any notable pain, and the pulsation diminished, the swelling practically disappeared, and there was apparently almost **complete cure.**

J. Buchholz[4] reports a case of aneurysm of the abdominal aorta in a woman of 35, in which improvement, which was practically a cure, occurred after administering *per os* a 10% solution of gelatin.

Scherwinsky[5] reports 4 cases of aneurysm of the aorta in which he used gelatin injections without being able to observe any influence.

**Thrombosis and Embolism.**—T. B. Futcher[6] describes briefly 3 interesting cases of thrombosis and embolism. In the first there was association of mitral stenosis and thrombosis of the left external jugular, subclavian, and axillary veins, with a subsequent embolism of the left popliteal artery. In another case thrombosis of the left femoral vein occurred in pulmonary tuberculosis—a not very uncommon event. In the third case there was thrombosis of the right axillary and brachial veins during malarial nephritis, the thrombosis probably being due either to the nephritis or to the anemia.

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[1] Lancet, Oct. 28, 1899.

[2] Jour. Am. Med. Assoc., Jan. 27, 1900.

[3] Klin. ther. Woch., No. 3, 1899.

[4] Norsk Magazine for Lagevidenskaben, No. 2, 1900.

[5] Therap. de Gergenwart, No. 12, 1899.

[6] Johns Hopkins Hosp. Bull., Feb., 1900, p. 48.

# DISEASES OF THE RESPIRATORY TRACT.

## METHODS OF EXAMINATION.

P. H. Pye-Smith [1] speaks with emphatic disapproval of many of **the terms used for obscure physical signs** in the chest. He first insists that physical signs do not point to definite pathologic processes, and one has no right to speak of "pneumonic" crepitation or "bronchitic" rales, and it is improper to speak of "Skodaic" resonance, "tympanic dullness," etc. Percussion-notes should be defined only by their variations from the normal in length, intensity, and tone, and not by indefinite names or fanciful terms. Harsh breathing is a term which he thinks ought to be excluded, as it is not in any sense definite; one should say merely loud breathing. He teaches that the vesicular murmur is due to the fluid vein in the glottis, modified by spongy lung, while bronchial breathing is the same murmur modified by and transmitted through solid lung. The terms moist and dry he considers inappropriate for rales, as he can not imagine such a thing as dry rales with free expectoration. The most important characteristic of rales is whether they are consonant or nonconsonant: that is, as to whether they have musical qualities or are mere noises. Consonant rales, in his experience, indicate lobular pneumonia. The condition called capillary bronchitis means also that there is a bronchopneumonia; in children, a lobular pneumonia with collapse, and in aged patients lobular pneumonia. In the intermediate period of life it usually indicates a secondary complication of miliary tuberculosis or lobar pneumonia or phthisis. He insists that in phthisis we are far too apt to look for definite signs, such as absolute dullness, consonant rales, cracked-pot sound, and the like. He feels convinced that isolated lobular pneumonia has little influence upon the percussion-note. The most common cause of dullness under the clavicle is thickening of the pleura rather than consolidation of the lungs.

C. F. Hoover [2] contributes an interesting paper on the **diagnostic value of the Wintrich tracheal sound.** Wintrich described the difference in the percussion-note when the mouth is open and when closed as characteristic of cavities in the lung. It has been shown, however, that it occurs from consolidated areas at the apices, and it may be produced by percussion over a consolidated base if the bronchi are not occluded; it may be of value in this way in differentiating between pleural effusion and consolidation of the lung. Hoover has never seen the sign with air or fluid in the pleura, but has observed it in lobar pneumonia and in tuberculosis. Percussion over the upper part of the sternum in normal chests does not give the Wintrich change of note when the mouth is opened and closed; but if the medium between the sternum and the trachea become of increased density, as from the presence of a tumor or an aneurysm, the change of note may be observed. Hoover has seen it in instances of mediastinal tumor repeatedly. It is also present in aneurysm and in pericarditis with effusion if there is sufficient fluid to cause distention of the culdesac of the pericardium over the origin of the large vessels at the base of the heart. It is important to observe several precautions in eliciting the sign. The patient should be told to elevate the chin, to take a deep breath, and to continue the inspiration after the full extent of thoracic expansion has been reached in order to keep the vocal cords apart. The mouth should be held open and the tongue protruded while the examiner is percussing over the manubrium, the percussion being continued throughout inspiration and the ear of the examiner being held directly in front of the patient's mouth. The two notes are due to the direct percussion and to the vibration of the air-column in the trachea. The tracheal tone is recognized by its peculiar metallic resonance, and by the fact that it is heard at the mouth.

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[1] Lancet, April 7, 1900.

[2] Am. Jour. Med. Sci., Oct., 1899.

E. Weisz [1] describes a **new method of physical examination** of the chest. He has observed that when patients phonate there is a peculiar forward bulging in the interspaces over the whole area which covers the lungs. Though the method has not yet been sufficiently tested, he thinks that it is probable that it will prove to be valuable in determining the borders of the lungs and of the adjacent solid organs. The examination is best made in daylight, with moderately strong light falling upon one side of the patient. He should be told to articulate short words beginning with the letter *k* or *d*, and each word should be spoken slowly, so that there may be no disturbance of respiration, since the respiratory changes in the interspaces would otherwise confuse the results. No elaborate study of the method in pathologic cases has as yet been made. Weisz has, however, observed in several cases of pleurisy that the sign was present. The vibrations were probably carried through the fluid. At times the projection may be observed over the stomach when this organ is distended, but it is not observed when the stomach is empty.

W. Ewart [2] gives an extended consideration of the **value of dorsal percussion,** especially of percussion of the spine. He describes the areas of dullness which are normally found in the back, and states that dorsal percussion constitutes an important and valuable means of determining the condition of the individual vertebræ and the presence of pus or of solid deposit. The dullness about the fifth dorsal spine is due to the loss of the resonant note of the trachea and to the replacement of this by a dulling influence, due particularly to the intratracheal glands together with other mediastinal structures. He attributes the dullness, however, chiefly to the normal intratracheal glands. He believes that a dullness about the left auricle may be easily and accurately outlined by percussion of the back. Minute descriptions are given of the areas of dullness normally found in the back.

A. Abrams [3] describes what he calls the **lung reflex,** which is produced by irritating the skin over the lung by means of cold friction or the faradic current. Dilation of the lung is stated to ensue, and may be recognized by diminished respiratory excursion, by obliteration of the apex-beat and of the cardiac and splenic areas of dullness, and by the appearance of hyperresonance on percussion; the lungs also appear brighter when examined by the x-rays. Abrams has used this lung reflex in the diagnosis between consolidation and atelectasis. When atelectasis is present, strong irritation will cause dullness to disappear. He thinks that in the bronchopneumonias of children dullness is chiefly due to atelectasis. He considers that such reaction to irritation indicates the value of active irritation of the skin by friction or cold water in furthering development of the lungs.

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[1] Deut. med. Woch., Mar. 1, 1900.

[2] Lancet, July 29, 1899.

[3] N. Y. Med. Jour., Jan. 13, 1900.

Aufrecht[1] insists upon the value of **percussing** the lungs both **during inspiration and expiration** to determine the alteration produced by respiration. He considers it particularly important for determining the presence of involvement of the apex of the lung and differentiating between pleural exudate and consolidation.

F. H. Williams,[2] in considering the **diagnostic value of the x-rays** in medical practice, believes that their greatest value is in studying diseases of the thorax. Pleuritic effusions may be distinguished from thickened pleura by noticing that if there is no fluid present the outline of the diaphragm is clearly seen. · He believes that changes in the central portion of the lung are much more readily determined in this way than by percussion. He insists again upon the value of the x-rays in diagnosing tuberculosis. In examining the abdomen one may see the liver, spleen, and left kidney, and may often determine the presence of calculi in the kidney. By administering bismuth subnitrate the outline of the stomach may be determined, or the stomach and intestines may be distended with gas and thus their outlines made to appear as bright areas.

## DISEASES OF THE BRONCHI.

J. Gordon[3] reports the case of a boy 4½ years old who had bronchitis with hard frequent cough; during one of the paroxysms swelling appeared on the right of the neck, rapidly involved the right side of the neck and cheek, and advanced downward to the lower border of the ribs anteriorly and posteriorly. There was also some swelling of the left side of the thorax above the nipple anteriorly. The swelling crepitated, and evidently was a **subcutaneous emphysema,** which Gordon believes was due to rupture of an air vesicle with infiltration of the intravascular tissue which advanced to the root of the lung, then to the mediastinum, and then along the fascia into the neck. The child had a temperature of 103° F. He improved rapidly, and the whole appearance of the emphysema had disappeared on the tenth day.

A. W. Dunning[4] describes a case of subcutaneous emphysema which is interesting because of the fact that it **occurred during** an attack of **vomiting.** During the strain accompanying this act the woman noticed a severe pain in the left side of the neck, which was followed by extensive emphysematous swelling, which, however, soon disappeared.

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[1] Deut. Arch. f. klin. Med., Feb. 6, 1900.

[2] Jour. Am. Med. Assoc., Nov. 11, 1899.

[3] Lancet, Dec. 9, 1899.

[4] Med. News, Aug. 26, 1899.

Teichmüller [1] describes the characteristics of **eosinophilic bronchitis.** The exact nature of the disease and its causes are not known, but it seems to be related to tuberculosis and also to a general neurotic family history. It is not uncommonly associated with rheumatism, and is likely to follow exposure. It commonly runs a subacute or a chronic course. Among the symptoms are dyspnea, frequent cough, mucoid expectoration containing a large number of eosinophile cells, some pain in the chest, disturbance of the gastro-intestinal tract, usually no fever. It is often difficult to distinguish the disease from tuberculosis, but the prognosis is good. The treatment which he chiefly recommends is hygienic and dietetic, giving potassium iodid if syphilis is suspected. [Our own examinations of sputum would indicate that such a form of bronchitis has no distinct existence.]

L. Rénon and L. Devillers [2] describe a case of chronic **pseudomembranous bronchitis** due to infection with **Aspergillus fumigatus.** It occurred in a woman of 39, whose work was sorting grains in order to select those suitable for planting in unfertile soil. Her work was carried on in a place which was so damp that the grain sprouted while lying in the sacks. She had pain in the mouth in 1894, and repeated attacks up to 1898. At this time she expectorated some membrane during an attack of bronchitis; new membranes were expectorated at almost every menstrual period. They were of whitish color with a tinge of green, and did not appear stratified. Microscopically, they were seen to be composed chiefly of mycelium and of spores of the aspergillus. Cultures proved virulent, the animals dying rapidly of experimental aspergillosis. Treatment had little effect.

G. H. Parry [3] describes a case of **bronchiectasis** in which vapor baths of guaiacol caused rapid improvement.

**Pneumonia.**—E. Palier [4] discusses **atypical forms of pneumonia,** and describes a case, terming it gastric pneumonia, in which the symptoms closely resembled those of appendicitis; abdominal section was performed by a consulting surgeon under the belief that disease of the appendix would be found. This, however, proved to be an error, and the autopsy of the case disclosed pneumonia associated with an empyema. Palier believes that abdominal symptoms in these cases are to be ascribed to disturbance of the pneumogastric center through the altered respiratory interchange, and he thinks that the abdominal pain is analogous to the pain felt in the knee in hip-joint disease.

Aufrecht [5] describes the case of a boy of 19 who was struck on the chest while swimming. He lost consciousness and sank to the bottom, but was brought to the surface after 4 minutes and was found to have some dullness over the right lung posteriorly the next day. The temperature rose on the following day, and a portion of the right lung also was found to be consolidated and the sputum was slightly hemorrhagic. The boy died on the sixth day. The autopsy showed consolidation of the whole right lung. Aufrecht believes that the pneumonia followed the injury, since the whole course was that of a **traumatic pneumonia.**

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[1] Deut. Arch. f. klin. Med., Aug. 18, 1899.

[2] Gaz. des Hôp., Dec. 1, 1899.

[3] Lancet, July 22, 1899.

[4] N. Y. Med. Jour., Sept. 16, 1899.

[5] Deut. Arch. f. klin. Med., Feb. 6, 1900.

Aufrecht [1] describes the case of a woman of 39 who, 17 days after a miscarriage, was admitted showing pneumonia of the lower part of the right lung. She died 4 days later, and the right lower lobe was found consolidated, but the surface was not granular. A **diphtheric membrane** was found in the uterus, and smear preparations from this and from the lung showed a large bacillus, which was apparently the **bacillus of Friedländer.**

Rispal [2] describes an instance in which considerable fragments of pulmonary tissue were expectorated, with the occurrence of marked hemoptysis. There were signs of consolidation of one lung, and a microscopic examination appeared to demonstrate that it was a case of cancer, but autopsy showed that it was one of the rare cases of **ulcerating chronic pneumonia** described in 1860 by Charcot.

R. B. Preble,[3] in discussing the **complications of pneumonia,** first directs attention to the plugging of the larger bronchi with fibrinous exudate. This gives rise to symptoms closely resembling pleurisy in many cases, but the pressure signs of pleurisy are generally absent. Cardiac complications are very likely to occur in such cases. Irregularity of the pulse is a sign of importance in pneumonia as indicating a tendency to cardiac failure. Also, accentuation of the second pulmonary sound during the course of the disease should arouse suspicion of cardiac failure. The same is true of the gallop rhythm over the heart. In speaking of meningitis he gives a collection of records of 16,333 cases of pneumonia which showed 64 cases of meningitis; but autopsy records show a much larger percentage, the figures ranging from 4.2% to 7.7%. The difference between clinical and pathologic records is explained by the fact that clinically the cases often run a latent course. In discussing arthritis as a complication he notes that it is rare, and that when it occurs it is usually monarticular. Joint pains, however, are common.

J. E. Tally [4] describes a case of pneumonia in a man of 76 in which on the seventh day there was **swelling of the parotid,** which increased rapidly, and on the tenth day suppuration was ascertained to be present. After spontaneous evacuation of the pus the swelling rapidly decreased, and the patient recovered entirely. Tally has found only 4 such cases recorded in English literature in the last 10 years.

J. Mandl [5] describes a case of croupous pneumonia in which, on the fourteenth day, and when convalescence was about established, a **keratitis** developed and advanced to perforation of the cornea. Cultures showed the presence of the pneumococcus in the ulcer, and the keratitis was attributed to the pneumococcus.

**Treatment.**—Broadbent,[6] in a general discussion of the treatment of pneumonia, directs attention to the value of **venesection** under special circumstances. When there is great engorgement of the right ventricle of the heart, with marked cyanosis, severe dyspnea and anxious expression, and a violently beating heart, with small pulse, the result of venesection is most satisfactory.

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[1] Deut. Arch. f. klin. Med., Feb. 6, 1900.

[2] V Congres français de méd. interne, 1899.

[3] Chicago Med. Recorder, Sept., 1899.

[4] Phila. Med. Jour., Mar. 24, 1900.

[5] Wien. med. Woch., Oct. 7, 1899.

[6] Practitioner, Jan., 1900.

C. A. Penrose [1] reports several cases of pneumonia which he treated by the combined use of **oxygen inhalations,** given by a special device, and **infusion of salt solution.** Three cases which were apparently hopeless were treated in this way, and one recovered. Penrose believes that the combination is especially valuable.

C. E. Elfstrom [2] reports 3 cases of croupous pneumonia in which he used his **heated blood treatment** with favorable results. In all, he has recorded 9 cases, only 2 of which were fatal, and he believes that these cases were hopeless before being injected, and should not be considered in calculating the mortality.

Cacacisinga [3] recommends the use of **silver salts** in the treatment of pneumonia. He describes 60 cases with only 3 deaths. He thinks that the results were strikingly satisfactory.

Reynaud [4] describes successful results from the treatment of grave cases of pulmonary infection, abstracting from 150 gm. to 300 gm. of blood, and then injecting subcutaneously 200 cc. to 600 cc. of **artificial serum.** The treatment was used in 60 cases with very satisfactory results.

**Asthma and Emphysema.**—A. Fraenkel [5] describes an extremely interesting case of bronchial asthma in which death occurred from increase of cardiac weakness. The autopsy showed changes which he considers sufficient to demonstrate the **origin of Curschmann's spirals.** He has previously reported observations upon these spirals in microscopic sections of the bronchi, but was in doubt as to the nature of the substance composing them. He now reports that a modification of the Biondi-Heidenhain triacid stain has convinced him that they are entirely composed of mucus. Histologic investigation of the case showed changes which consisted essentially in curious elongation and narrowing of the cells, so that they came to be long strands, and attached to them was an extremely adherent substance, which on staining proved to be mucus. Various stages could be observed in the formation of the spirals, so that Fraenkel was convinced that their formation occurred by elongation of the cells and excretion of considerable quantities of mucus, which adhered, and, by the movements of air in expiration, was whipped about until it came to assume a spiral form around the elongated cell. The nature of the central portion of the spiral has been in doubt. This Fraenkel considers is the remnant of the original cell. He observed many eosinophiles in the bronchial exudate. These were, as has been noted before, chiefly mononuclear. He believes that these eosinophile cells come from the blood; he was able to observe entirely similar cells in the small vessels in the neighborhood of the bronchi, and the eosinophile cells were seen apparently making their way between the epithelial cells of the bronchi.

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[1] Johns Hopkins Hosp. Bull., July, 1899.

[2] N. Y. Med. Jour., Sept. 30, 1899.

[3] Bull. dell. Assoc. Sanitat., An. I, N. 9.

[4] Gaz. hebdom. de méd. et de chir., Nov. 9, 1899, p. 1077.

[5] Deut. med. Woch., April 26, 1900.

F. Riegel[1] believes that **all cases of asthma,** whatever their exciting cause, are **essentially nervous,** and that the attack is produced by the reflex or direct irritation of the vagus nerve. Hence he considers that the most rational treatment is the use of atropin, and he substantiates the statements of v. Noorden concerning the value of this drug; when given subcutaneously in doses of from ½ mg. to 1 mg., he thinks that it practically always controls the attack, the dyspnea vanishing and the distention of the lungs rapidly diminishing. He has as yet no experience to relate concerning the recurrence of attacks after this treatment.

W. B. Bell[2] believes that asthma is caused by **active dilation of the pulmonary vessels.** He believes that the pulmonary vessels dilate actively and not merely passively. His theory of the causation of asthma is based chiefly on the effects of drugs, and especially upon the action of ergot, which he has found to be very beneficial in asthma.

S. Solis-Cohen,[3] in discussing the use of **adrenal extract in asthma,** states that in spasmodic cases it has little influence, but in cases in which the attack is the result of vasomotor disturbances it maintains vascular tone, and has a valuable influence. He gives from 5 to 10 grains every 2 or 3 hours. Moderate doses should be used at first.

Campbell,[4] in discussing the treatment of pulmonary emphysema, states that we should endeavor to preserve the elasticity of the lungs, to prevent the overaction of the costal elevators, and to prevent fixation of the chest. To this end we should, as far as possible, limit cough, straining, and dyspneic respiratory efforts. At the same time the patient should be given expiratory exercise. Campbell discusses an instrument devised by Willock which he considers extremely valuable as an aid in exercises directed toward overcoming inspiratory fixation of the chest. The use of this, together with well-directed muscle-exercises, will, he believes, do much to improve emphysema, particularly when treatment is begun early.

M. Manges,[5] in concluding an article on the therapeutics of **heroin,** gives the results of a collective investigation of the opinion of the profession concerning the drug. Of 141 persons who replied to letters, only 4 expressed themselves unfavorably. He makes a report of 416 cases treated, of which 75 were his own; in only 21 were there any unfavorable effects from the heroin. He considers it the most valuable drug that we have in emphysema and asthma. During asthmatic attacks it should be given subcutaneously in the form of the hydrochlorate. In 2 cases he used it with success in the treatment of the morphin habit. [It must not be forgotten, however, that heroin is, after all, a derivative of morphin. The same objections apply to it as to other opium derivatives, though the danger of heroin is comparatively insignificant. We do not agree with Harnack, though a word of warning is deserved.]

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[1] Deut. med. Woch., Oct. 12, 1899.

[2] Edinb. Med. Jour., Oct., 1899.

[3] Jour. Am. Med. Assoc., May 12, 1900.

[4] Brit. Med. Jour., Oct. 28, 1899.

[5] N. Y. Med. Jour., Jan. 20, 1900.

Harnack [1] believes that heroin has been used with dangerous freedom, and considers that it is a more dangerous drug than morphin, and that its free use is likely to lead to addiction to the drug. Draeser [2] considers that Harnack exaggerates the toxicity of heroin.

H. D. Fulton [3] recommends heroin for severe cough in influenza, phthisis, bronchitis, and other similar affections, and states that the hypodermic use of the drug may be facilitated by dissolving it in acetic acid.

Herwisch [4] has had valuable results in the treatment of cough with heroin, and finds that it does not tend to cause headache or disturbance of the stomach, and that there is little tendency to resulting constipation or drowsiness.

Wierzbicki,[5] after a considerable number of experiments with the use of heroin, recommends it for cough, and states that it has no influence upon temperature or upon the circulatory or digestive organs, and that the only unpleasant effect is a slight irritation and dryness of the throat.

Klink [6] reports 2 cases in which accidentally about $\frac{5}{6}$ of a grain of heroin was taken 3 times a day without harmful results.

T. Janisch [7] reports upon the use of **dionin** (the hydrochlorate of ethylmorphin) in the treatment of disease of the respiratory tract. He has had satisfactory results from its use for the relief of cough and night-sweats. It does not interfere with expectoration and it increases the volume of respiration. The drug is a powder, of slightly bitter taste, and is readily soluble in water, alcohol, or syrup.

## MISCELLANEOUS AFFECTIONS OF THE LUNGS.

**Foreign Body.**—G. Killian [8] reports the case of a man of 42 who aspirated into his lung in March, 1896, a **piece of bone,** and who since that time had suffered continuously more or less distress, chiefly consisting of purulent expectoration, tendency to febrile attacks, and depression of the general health. He had some dullness over the central portion of the right lung and there were rales at the base. At the left apex there were signs which gave rise to suspicion of tuberculosis, though no bacilli were found. Direct bronchoscopy showed the presence of a foreign body in the right bronchus at its entrance. It was necessary to introduce the bronchoscope between the back teeth and to have the head turned toward the side in order to see the mass. Killian removed a piece of bone 15 mm. long by 11 mm. broad, relying upon the sense of touch alone and grasping the mass with forceps. The man recovered almost complete health.

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[1] Münch. med. Woch., July 4, 1899.

[2] Münch. med. Woch., July 25, 1899.

[3] N. Y. Med. Jour., Dec. 30, 1899.

[4] Therap. Gaz., Nov. 15, 1899.

[5] Klin. therap. Woch., No. 27, 1899.

[6] Münch. med. Woch., Oct. 17, 1899.

[7] Münch. med. Woch., Dec. 19, 1899.

[8] Deut. med. Woch., Mar. 8, 1900.

**Pneumoconiosis.**—Bäumler,[1] in discussing the diagnosis of **changes** in the lung **caused by inhalations of dust,** states that the most important are a result of the retraction of the lung. One of the most valuable points is retraction of the anterior margin of one or both lungs in the upper portion; this leaves an abnormally large area of imperfect resonance or dullness in the central portion of the chest anteriorly. The symptoms produced are commonly cough, with expectoration, and with the evidences of thickening of one or both apexes, and of bronchial catarrh with little or no fever. As a rule, the general nutrition and circulation suffer. The patients are commonly men of middle life or older. The cases are not infrequently complicated with tuberculosis. Bäumler states that tuberculosis itself may be favorably influenced at times by inhalation of dust, owing to the fibroid changes produced. [We have found the breathing of a peculiar harsh character in cases of pneumoconiosis. This has seemed to us to result from the thickened condition of the bronchial tubes and the general pulmonary induration.]

W. W. Betts[2] reports his study of 30 cases of **chalcicosis pulmonum** which occurred in employees of a quartz-crushing mill, and which resulted from the inhalation of dust. Many of the workers in this mill died within less than a year, and almost all of them became ill. Within a few weeks after beginning work the mucous membranes become irritated, bronchitis develops, and the men lose weight and become dyspneic. They survive about 10 months on an average. In the latter part of the disease they have decided fever. Postmortem examination demonstrates cirrhosis of the lungs, sometimes multiple abscesses, breaking-down of tissue, and infiltration with dust. Betts condemns the operators ot such mills for their lack of care concerning their employees, and considers that lives are uselessly sacrificed by not providing proper fans and dust collectors.

G. Carriere[3] describes a case of pneumoconiosis with extensive infiltration of one lung which occurred in a worker in a cocoa factory. The man was constantly surrounded by the dust of cocoa, and it was found that his sputum contained large quantities of the cells of this plant, and was of a color resembling that of cocoa. He ultimately recovered entirely. The **condition** was believed to have been **produced by the cocoa.**

Hermann[4] describes 6 cases of **carcinoma of the lung.** He considers diagnosis during life, as a rule, very difficult or impossible. It is sometimes established by finding characteristic particles of malignant growth in the sputum or in the pleural exudate. Hemorrhagic exudate is also of importance, particularly if the exudate is at first serous and afterward hemorrhagic. At times there is fibrous pleurisy and retraction of the chest. As a rule, the pleurisy is exudative, occasionally purulent. The discovery of metastases in the axillary glands or in those about the clavicle is a very important sign. The sputum may resemble that of pneumonia, or may at times be somewhat like that of gangrene of the lung. Pressure symptoms are very common. Often it is extremely difficult to differentiate the condition from tuberculosis of the lungs.

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[1] Münch. med. Woch., April 17, 1900.

[2] Jour. Am. Med. Assoc., Jan. 13, 1900.

[3] Gaz. hebdom. de méd. et de chir., Nov. 2, 1899.

[4] Deut. Arch. f. klin. Med., Aug. 18, 1899.

## DISEASES OF THE PLEURA.

Dieulafoy [1] discusses **appendicial pleurisy,** using as the chief basis of his paper a case occurring in a young man in which the symptoms of appendicitis were followed by pleurisy, and the man ultimately died of a fetid pleurisy. Postmortem examination showed a collection of pus about the appendix, with pus tracts leading upward to the pleura. There was a foul right-sided pleurisy with an embolic abscess in the left lung. Bacteriologic investigation showed the presence of the colon bacillus and of a proteus form, as well as some micrococci in the pus about the appendix and the pleural fluid. It was evident that the appendicial trouble had given rise to the affection of the pleural cavity, and had caused death in this way. A series of further observations of the same kind are detailed, and the conclusions at which Dieulafoy arrives are that pleurisy may be the result of infection consecutive to appendicitis, and that there is a distinct appendicial form of pleurisy. There is often first a subdiaphragmatic collection of pus, the thoracic cavity then becoming involved. The pleurisy usually appears only after some days have passed subsequent to the development of the appendicitis. It always occurs on the right side. Usually the pleurisy is fetid, but it may be a dry form or may be accompanied by a serous effusion. The prognosis is very serious. In the presence of right-sided pleurisy with fetid effusion an appendicial origin should always be taken into consideration; and if the diagnosis is established, the pleurisy and the appendicitis should be operated upon simultaneously. [We recall one case of suppurative perityphlitis with upward extension behind the colon. A subdiaphragmatic abscess and suppurative infection of the liver resulted from this extension, and the process involved the diaphragm, though it had not penetrated to the pleura. This form of extension may, of course, occur, but it is hardly judicious to speak of the resulting pleurisy as a distinct variety. Excessive classification of pathologic conditions is unwise. A suppurative inflammation of the pleura may occur in various ways, and careful clinicians will consider all possibilities in searching out the original focus of disease in any given case.]

R. Bernard [2] describes a case to illustrate the belief which he expresses that the **appearance of fibrin** in pleural effusions is a **favorable sign,** and one that indicates that the fluid will probably not reaccumulate, and that the case will subsequently run a favorable course.

Carageorgiadès [3] states that he has practically always observed the occurrence of **urticarial eruption** with the beginning of absorption in cases of exudative pleurisy. He attributes this to intoxication of the organism with the toxins present in the pleural exudate, and believes it a very favorable sign, indicating the early absorption of the exudate.

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[1] Bull. Acad. de méd., April 10, 1900.

[2] Gaz. hebdom. de méd. et de chir., Oct. 1, 1899.

[3] V Congres français de méd. interne, 1899.

W. S. Cheesman and W. S. Ely [1] report a **remarkable case of hemorrhagic effusions** into the pleuræ and peritoneum in a woman of 47. The affection began in 1890 with pleural effusion. The patient was tapped and a bloody fluid withdrawn, and by January, 1892, the right pleura had been tapped 19 times, a bloody fluid being withdrawn at every tapping. In order to obliterate the right pleural sac it was washed out, and iodin solution was introduced and withdrawn. After this had been done a second time success was secured, but bloody effusion appeared on the left, and was aspirated 7 times within 3 months. The disease on this side was then overcome by iodin injections. Afterward a hemorrhagic effusion appeared in the peritoneum, and was tapped 43 times in 5 years, large amounts of bloody fluid being withdrawn each time. The patient had a large fibroid tumor, and she lost considerable blood from menorrhagia also. After cessation of menstruation with the climacteric, the accumulation of fluid ceased. Tuberculosis was excluded and the history excludes malignant disease, and the nature of the affection remains in doubt. The authors are inclined to consider it primary hemorrhagic effusion.

G. G. Sears [2] reports an interesting case of acute **hemorrhagic pleurisy** which followed exposure. The patient's red blood-corpuscles were found reduced below 3,000,000, and tapping of the pleura withdrew 8 ounces of blood. Larger quantities were removed each time in 7 subsequent tappings. Ultimately a portion of the ninth rib was removed and the pleura was drained, 2 pints of red fluid escaping. The drainage-tube was left in, and the patient recovered within 2 months with only a small sinus. Tuberculosis was supposed to have caused the pleurisy.

E. S. Boland [3] describes a case of **pneumothorax** in which the cause was obscure. The attack came on in the night with sharp pain in the chest, and physical examination the next morning showed the presence of a pneumothorax; fluid accumulated, which upon aspiration proved to be almost pure blood. Two subsequent aspirations were undertaken, 73 ounces in all being removed. The patient subsequently improved, and ultimately recovered entirely.

**Treatment.**—C. H. Lewis [4] advises attempting to **secure a deposit of fibrin** on the pleural surface, and the absorption of the serous exudate by withdrawing some serum from the cavity, dissolving this in methylene-blue, and then reinjecting the solution. The aspirated serum should be withdrawn into a vessel containing methylene-blue, the two then being mixed with a glass rod and reintroduced. The vessel is kept covered during the process, and is also previously thoroughly sterilized in order to prevent infection. Twenty cases treated by this method are discussed. The results were considered very satisfactory. The injection is not painful. Lewis believes that the majority of cases of pleurisy are tuberculous, and that methylene-blue is inhibitory to the activity of the tubercle bacillus, as also to other micro-organisms. The treatment in the 20 cases which he reports averaged about 13 days in duration. [We can not indorse this or other methods of treatment in which various solutions of chemical substances are injected into the pleural cavity. Neither can we agree that most cases of pleurisy are tuberculous. Careful investigations have disproved this view. Medicinal treatment, rest, and simple tapping cure most cases; injections into the pleura are not free from certain dangers.]

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[1] Am. Jour. Med. Sci., Aug., 1899.

[2] Boston M. and S. Jour., Oct. 19, 1899.

[3] Boston M. and S. Jour., Mar. 29, 1900.

[4] Med. Rec., Dec. 30, 1899.

H. Ortner,[1] in discussing the **complications** which occur **during thoracentesis,** directs especial attention to acute pulmonary edema, and reports 2 cases. In the first case there was expectoration of albuminous material and a paradoxic pulse after the tapping. The autopsy showed a pericardial concretion, a mediastinitis which involved the phrenic nerve, and advanced myocardial change. In the second case albuminous expectoration was observed also, and there was a fibrinous coagulum. The patient died afterward with tuberculosis. Autopsy showed tuberculous pericarditis, with brown atrophy of the heart, and bilateral pleurisy. The occurrence of pulmonary edema under such circumstances Ortner attributes to the abnormal permeability of the blood-vessels of the lung, and also to the immobility of the contents of the mediastinum resulting from the mediastino-pericarditis, the lung being unable to expand. It is important, before undertaking aspiration of the pleural cavity, to determine that the heart is in fairly good condition. It is also possible and necessary to determine that the contents of the mediastinum are movable. Evacuation should be undertaken slowly and the heart should be stimulated previously.

H. Hellandall [2] describes **an attachment for a trocar** which he has devised to prevent the entrance of air into serous cavities when aspirating them. The essential point in the instrument is a valve which allows the passage of fluids outward, but at the moment when there is any negative pressure in the cavity, the valve closes and prevents the entrance of air. He recommends it especially in removing the fluid in ascites, and found it quite as valuable as the aspiration apparatus in pleural effusion. He states that one may entirely empty cavities without danger of admitting air into them.

**Echinococcus.**—L. Rénon [3] describes a case of multilocular echinococcus of the **pleura and right lung** in a man of 36. This, he states, is the first instance of this disease in man which has been observed in France.

**Endothelioma.**—S. Vellinghausen [4] reports a case of primary endothelioma of the pleura in a man of 43. He considers irregularity in the line of dullness of great importance in the diagnosis of pleural growths. In the hemorrhagic effusion in this case he found numerous round polymorphomononuclear and multinuclear cells and large lymphoid cells.

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[1] Wien. klin. Woch., Nov. 12, 1899.

[2] Deut. med. Woch., April 26, 1900.

[3] Compt. rend. de la Soc. de Biol., Feb. 17, 1900.

[4] Münch. med. Woch. May 8, 1900.

## DISEASES OF THE MOUTH AND PHARYNX.

M. Cohn [1] reports some results from the **investigation of the saliva.** He found many indicators useless in measuring the alkalinity because of the large amount of $CO_2$ in the saliva. Methyl-orange proved to be the most satisfactory, and this gave reliable results. The average alkalinity of the saliva corresponded to a solution of sodium hydrate of a strength of 0.0154 %, though it varied considerably. The figures given by earlier authors for the alkalinity he considers have usually been too high. The alkalinity was found greatest in the morning when the stomach was empty; it decreased in the forenoon, increased again at noon, and reached its highest point at this stage. It then decreased again in the afternoon, and increased toward the time of the evening meal. He never found the reaction acid in normal persons. He directs attention, however, to the fact that the saliva has frequently been found acid in infants and in adults who are the subjects of diabetes or of diseases of the mouth, esophagus, or stomach. He was not able to observe any relation between the acidity of the stomach-contents and the alkalinity of the saliva. He found an unusually high degree of alkalinity in some cases of paralytic sialorrhea and in the saliva of persons who had received pilocarpin, and the freezing-point of the saliva was somewhat more reduced in these cases than it was in normal persons. A number of experimenters have attempted to cause absorption of blood exudates or of metabolism products in cases such as pleural effusion and in nephritis. Cohn has attempted the same thing, without definite result in most cases. He could not note any marked change in the alkalinity or in the molecular concentration, though the amount of chlorids seemed to be increased. In 2 cases of chronic parenchymatous nephritis, however, he found the molecular concentration high as indicated by the lowering of the freezing-point. He states that he has often been able to determine a distinct increase in the molecular concentration of the blood in uremic cases. He did not, however, observe any increase in the amount of saliva secreted in uremia. He continues his article by giving his results after the administration of test-meals in the usual manner and through a tube without chewing. It was always found after taking food through a tube that the acidity of the gastric contents was diminished. The motility was found little influenced by the manner of taking the meal, though the quantity found in the stomach after introducing the meal through the tube was always somewhat greater than when taken in the normal manner. [This confirms the results of others, and adds to the evidence of the importance of the saliva, the act of chewing, and the stimulation of the sense of taste in exciting the functions of the stomach.]

F. P. Weber [2] describes the case of a middle-aged man who since boyhood had been subject to **paroxysmal attacks of salivation** accompanied by a general feeling of distress. The attacks were usually induced by exercise after a full meal, and could practically always be prevented by a little care in the avoidance of large meals or by lying down after a meal. He had some atony of the stomach, and the affection was believed to be due to dragging the stomach downward by overloading it, and thus reflexly stimulating the salivary glands to action. Reflex salivation occurs sometimes in pregnancy, in cirrhosis of the liver, and in other abdominal affections.

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[1] Deut. med. Woch., Jan. 25 and Feb. 1, 1900.

[2] Edinb. Med. Jour., April, 1900.

**Angina.**—Damany [1] describes an **epidemic of angina** which occurred in Rennes and persisted from September, 1898, until January, 1899. The disease presented the appearances of an acute herpetic angina, but showed intense general symptoms, and in many cases there were complications in other organs. Skin eruptions were frequent. In one case there was streptococcic septicemia, which caused death. Some of the cases in children had symptoms exactly similar to those commonly described as glandular fever. Bacteriologic examination in all these cases showed the presence of a **streptococcus,** which always took Gram's stain, sometimes clouded bouillon and sometimes did not, formed punctiform colonies on gelatin, and gave no apparent growth on potatoes. It showed a feeble virulence when injected into rabbits.

Siegert [2] observed an **epidemic of follicular tonsillitis** which affected 28 persons in Strasburg who lived near one another. He considers the disease readily transmissible, and thinks that cases should be isolated, and that children should not be sent to school for as long as 5 days after they have come in contact with a case. The incubation period seems to be about 4 days.

C. E. Edson [3] reports a case of septicemia originating in a severe angina and in which there was suppuration in the lungs which was treated by the **injection of normal salt solution.** The patient had lost ground rapidly up to the point of the first injection, but after this improved, and each of 3 subsequent injections caused a continued improvement. A cure was finally obtained.

## DISEASES OF THE ESOPHAGUS.

G. Muller [4] reports a very interesting observation of **fatal hemorrhage from esophageal varices** in which he found no evidence of cirrhosis of the liver, even upon microscopic examination, while the esophagus showed marked varices in the portion just above the cardia and in the upper portion of the esophagus beneath the pharynx. The liver showed marked fatty degeneration, which was attributed partly to the alcohol, but chiefly to the severe hemorrhage. The esophagus showed capillary hemorrhages in the mucous membrane, partial loss of the epithelium, and marked change in the vessels. These changes were attributed to the direct action of the alcohol, because of their corroded appearance, their situation, and the absence of cirrhosis of the liver. He believes that other cases which have been reported demonstrate that alcohol may produce phlebectasia by direct action. Letulle[1] describes a similar case in an inveterate alcoholic, 34 years old, who died after 4 severe hemorrhages. The autopsy showed that the liver was enlarged, but it was not sclerotic, and the portal vein was entirely free from disease. The esophagus showed numerous dilated veins, chiefly in the lower part of the canal. [The local effect of alcohol must undoubtedly be taken into account in studying the etiology of esophageal varicosities.]

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[1] Bull. Acad. de méd., Sept. 19, 1899.

[2] Münch. med. Woch., Nov. 21, 1899.

[3] Boston M. and S. Jour., July 27, 1899.

[4] Gaz. hebdom. de méd. et de chir., May 20, 1900.

J. S. Pyle[2] describes a case in which **stricture** of the esophagus came on **after typhoid fever.** There had been much dysphagia and pain during the course of the typhoid, and the pain and difficulty in swallowing were much increased when acids or acid foods were taken. The dysphagia had continued, and it was thought to have been a typhoid ulceration of the esophagus resulting in stricture. Pyle describes a method of treating the case which he adopted. The essential of the instrument which he used was a wire guide, over which was a tube having a finger-shaped silk sac at the end. The sac was passed part way through the stricture, and by means of a force-pump it was blown up with air so that it was distended on each side of the stricture. Subsequent to this, water was forced into the sac, and in this way dilation of the stricture is said to have been accomplished.

A. Fraenkel[3] describes 2 cases in which stenosis of the esophagus probably followed **peptic ulcers.** In the first the signs of stenosis of the esophagus followed symptoms of disordered digestion that had been present for a long period. Obstruction was found at the cardia. The man died after gastrostomy, and the autopsy showed a cicatricial stricture of the esophagus. The development had been gradual, and had probably followed ulcer of the esophagus. In the second case there had been severe abdominal pain and distention, with constipation, some hours before the attack. Three months before being seen the patient had experienced severe pain and vomiting after eating, and he had vomited coffee-ground material. He died, and an ulcer of the lower end of the esophagus and of the cardiac end of the stomach was found. Fraenkel reports a series of 20 cases of probable peptic ulcers of the esophagus which he has collected from the literature.

A. Glockner[4] describes a case of peptic ulcer of the esophagus in a man who had been treated for alcoholic gastritis for many years. The trouble began with dysphagia, and stricture was found 21 cm. from the teeth. The difficulty in swallowing disappeared, but after the patient's death investigation showed a **girdle-like ulcer** in the lower portion of the esophagus, with hypertrophy of the muscular wall, and also ulceration of the stomach and duodenum. The cause of the ulceration was thrombosis of the vessels, thrombi being discovered in several places.

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[1] Gaz. des Hôp., Dec. 24, 1899.

[2] Phila. Med. Jour., Feb. 3, 1900.

[3] Wien. klin. Woch., Oct. 19, 1899.

[4] Deut. Arch. f. klin. Med., vol. LXVI, Festschrift.

E. Fuerst[1] reports a **curious case of severe spasm** of the esophagus after the introduction of the stomach-tube. The patient was a hysteric woman of 36 who had shown symptoms suggesting cholelithiasis, and the stomach-tube had been introduced because of the severe vomiting, in an attempt to quiet this. No gastric contents flowed from the tube, and an attempt was made to draw it out. It was found, however, to be tightly caught, and could be moved neither way. An injection of morphin had no effect, and ultimately, after a half hour's manipulation, chloroform narcosis was used, and only then was it possible to move it. It was found that the tip of the tube was folded over itself, and probably this accident, associated with the spasm of the esophagus, led to the unusual difficulty in withdrawing it.

H. Stern[2] describes a case of **gouty spasm** of the esophagus. [The case occurred in a subject with a gouty family and personal history, but there seems to be no very definite reason for believing that the spasm was due directly to gout.]

A. Abrams[3] records the case of a woman with hysteric characteristics who had marked eructations. The study of the case with the fluoroscope showed no movement of the stomach during the belching, and the introduction of a stomach-tube into the esophagus caused the eructations to disappear. It was believed, therefore, that the air was **belched from the esophagus,** and not from the stomach. [This form of air-swallowing and eructation is not unknown. A remarkable case has been under our observation, in which the symptoms have persisted for a long time—several years. The patient, after a sort of modified swallowing, makes remarkable and often terrifying sounds as the swallowed air is discharged.]

V. Blum[4] describes a case of **diverticulum** of the esophagus in a man of 66, in which the symptoms had been chiefly signs of lodgment of the food at about the region of the larynx, and relief by pressure upon the right side; pressure also caused a gurgling sound and regurgitation of the food into the mouth. An obstruction was found 23 cm. from the teeth, and the introduction of a bismuth solution and examination with the x-rays showed an oval shadow indicating the presence of a diverticulum.

T. Rosenheim,[5] in discussing **atony and atonic ectasis** of the esophagus, insists that these conditions are often primary and often the cause of cardiospasm. Even though the cardiospasm seems to be the most prominent symptom in the case, disease of the mucous membrane in the ectatic area causes reflex irritation and the production of the spasm. Cases to illustrate the production of cardiospasm from a primary atony are reported in the paper. In the treatment of such conditions Rosenheim recommends giving nourishment through the stomach-tube 3 times a day in order to avoid irritation of the diseased mucous membrane, and then withdrawing the tube into the esophagus and applying a solution of silver nitrate alternately with borax solution. If this is not productive of good results, gastrostomy should be undertaken in order to give the esophagus complete rest for a time. Electricity may well be used also, and Rosenheim describes a special electrode for treatment of the esophagus. The important points in the diagnosis of atony of the esophagus are the fact that fluids are swallowed without any difficulty, while more solid substances cause distress, because they require muscular effort to carry them down; the sound is passed without any special difficulty, and the use of the esophagoscope shows marked folds of the mucous membrane in the lower third of the esophagus, which may be pressed away easily, and the end of the instrument may be moved about throughout an abnormally wide area. Atonic ectasis of the esophagus is not a simple matter, to be dismissed from thought carelessly, since it may cause death of itself or may dangerously reduce the nutrition of the patient, and sometimes may end in carcinoma. An instance of the latter result is reported. Rosenheim insists that the fact that for many years a patient has had symptoms largely attributable to the nervous system furnishes insufficient proof that the case is purely nervous, or is, at any rate, nonmalignant. He reports 2 cases in which there had been prolonged history of functional nervous disturbance, with difficulty in swallowing, both of which seemed to be pure nervous spasm of the esophagus. Examination of one with the esophagoscope showed carcinoma; in the other death ultimately occurred, and the stricture was found to be organic, though of doubtful nature.

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[1] Deut. med. Woch., April 5, 1900.

[2] Interstate Med. Jour., Aug., 1899.

[3] Phila. Med. Jour., Aug. 12, 1899.

[4] Wien. klin. Woch., Mar. 15, 1900.

[5] Deut. med. Woch., Nov. 9, 16, and 23, 1899.

F. A. R. Jung [1] contributes an interesting paper on the **diagnosis of diverticula in the lower part** of the esophagus, directing attention particularly to the distinction between idiopathic dilation of the lower part of the esophagus and diverticula. The method of Rumpel is one of the most satisfactory, consisting in the introduction into the stomach of a tube with a series of perforations, and another tube alongside of it into the dilated part of the esophagus. If water introduced into the second tube runs completely into the stomach, it indicates the presence of dilation. If much of the water can be siphoned out again from the second tube, it indicates a diverticulum. Jung's method was an elaboration of this, consisting in the introduction of a smaller tube into the stomach-tube, this smaller tube containing perforations only at the distal extremity. The advantages of this tube are that with it one may aspirate gastric juice and examine it, and that the stomach may be filled and emptied at will. The first tube is introduced, and then the small tube is inserted into it. One first introduces as much water as is comfortable through the small tube; none should return through this or through the larger tube until the stomach is filled to overflowing. Rumpel's test may then be carried out with the other tube, which is introduced into the esophagus. Another point showing the value of the second tube within the other is that it tells whether the perforated tube is in the stomach or in the esophagus. If water is readily returned at once through the perforated tube, it may be considered that this is not in the stomach, but in the esophagus. Jung reports 2 cases; one in a man, the other in a woman. The man had experienced marked difficulty in swallowing, which had been increasing, and attacks of extremely painful cramps in the pit of the stomach, with a sensation of choking. Food was ejected frequently, sometimes even during a meal; it was difficult to tell whether the food came from the stomach or from the esophagus. He had severe coughing attacks at night, which were relieved by vomiting. He had a sensation that the food did not enter the stomach. Food which had evidently been there for as long as two days was withdrawn from the esophagus. The stomach-contents showed free HCl and gave a reaction for peptones. The contents of the esophagus were slightly acid and gave no peptone reaction, there was no reaction for rennet ferment, and sugar reaction was strongly positive, as was that for lactic acid. The use of Jung's tubes showed that the condition was a diverticulum. This was confirmed by examination with the x-rays and with the gastrodiaphane. Treatment consisting chiefly of lavage of the esophagus had made the patient almost free from his distress. In this case as well as in the second the spasms of the cardia were much lessened by the administration of small seidlitz powders or other carbonic waters.

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[1] Am. Jour. Med. Sci., April, 1900.

W. Fleiner [1] discusses **dilations in the lower portion** of the esophagus under the headings of dilation above the diaphragm and those below the diaphragm, the latter being dilations of what Fleiner calls the cardiac antrum. He gives notes of 6 cases, in 3 of which there were apparently several spindle-shaped dilations. The diagnosis was made clinically in 4 cases, and the condition was found at autopsy in the other 2. He considers the dilation to be dependent upon congenital conditions. The symptoms are chiefly a feeling of pressure on the sternum, a necessity for waiting after food is swallowed until it passes into the stomach before more is taken, often pain from distention of the sac, frequently regurgitation, and sometimes rumination. There may be severe acute symptoms of obstruction, particularly at meals. The tube, when passed, shows an accumulation of food and fluid above the stomach. The use of the x-rays may help in diagnosis. Sometimes there is a history of difficulty in swallowing, beginning early in life. The treatment is chiefly washing out the sac with astringents or disinfectants in weak solutions, and feeding by the introduction of the tube into the stomach.

## DISEASES OF THE STOMACH.

### METHODS OF EXAMINATION.

Riegel,[2] in discussing the **tests of the secretory power** of the stomach, notes that while the test-breakfast is easier to use, it is sometimes necessary to employ a test-dinner also. The stomach may be capable of giving normal secretion after a test-breakfast when this does not take place after the test-dinner. Free HCl may be present with the former, but not with the latter, and it must be present or the secretion can not be considered normal. In uncommon cases a test-dinner may show an excess of HCl when the test-breakfast does not; this is probably an indication that the digestive organs react to strong stimuli when they do not react to weaker ones.

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[1] Münch. med. Woch., April 17 and 24, 1900.
[2] Münch. med. Woch., Nov. 7, 1899.

J. Troller[1] **criticizes Talma's method** for obtaining pure gastric juice. He states that the solution of Liebig's meat extract which Talma uses contains albuminous substances, and that therefore the whole amount of acidity found in the juice can not be considered free HCl, and that as one for obtaining pure gastric juice the method is faulty. Troller has investigated the effect of chewing lemon-rind and mustard, and has found that by this means gastric juice may be obtained without any direct irritation of the stomach. The mustard caused a much more active secretion than the lemon-rind. The mere chewing of bread or beefsteak, when these foods were not swallowed, caused an active gastric secretion also, and the amount secreted while chewing food was much greater than that after chewing either lemon or mustard; also beefsteak caused a much freer secretion than bread. In cases of hypochlorhydria the amount and the degree of acid obtained after chewing lemon, mustard, or foods decreased until finally no secretion was caused. Troller decided that the **first impulse to the secretion** of gastric juice is **caused by chewing,** and that thus digestion is set in motion. He also repeated Biemacki's experiments, administering finely divided bread through a tube at one time, and allowing the patient to chew bread at another time. He obtained the same results as others, secretion being much greater after the bread was chewed and swallowed. He believes that we should learn to distinguish between pathologic secretions due to changed reaction of the stomach toward direct irritation, and those changes in gastric secretion due to alterations in the reaction to reflex irritation produced by mastication. He further studied the direct irritative action of a number of substances when taken into the stomach, and found that of albumins, starch, olive oil, cane-sugar, and grape-sugar, albumins produced much the most marked effect; water caused some irritation, as did starch, but fat and grape-sugar caused a reduction of the secretion.

F. Winkler and C. Stein[2] used **iodipin in testing the function** of the stomach, choosing this preparation because it is a combination of iodin with fat and the iodin is not set free until the substance reaches the intestine and the fat undergoes a certain degree of digestion. The time of the occurrence of the iodin reaction in the sputum was noted after iodipin had been given, and 46 cases were thus studied. They think that the action should occur in from $\frac{1}{2}$ to $\frac{3}{4}$ of an hour after taking the iodipin, and that a delay of over one hour indicates disturbance of the motor power in the stomach. Delay of from 1 to 4 hours occurred in cases of gastroptosis, dilation, and carcinoma.

H. L. Hewes[3] describes as a **simple clinical method for the**

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[1] Zeit. f. klin. Med., Bd. XXXVIII, Hefte 1, 2, u. 3.

[2] Centralbl. f. innere Med., Aug. 19, 1899.

[3] Boston M. and S. Jour., Jan. 4, 1900.

**quantitative analysis** of the gastric contents the following: Free HCl is estimated by the dimethyl test of Töpfer, the final reaction being controlled by the use of tropæolin; the total free acids and acid salts are estimated by titrating with Congo paper; the total acidity is then estimated in the usual way. By subtracting the free HCl from the total free acid and acid salts one obtains the amount of total organic acids plus acid salts. Subtract the total free acids plus acid salts from the total acidity, and one obtains the total combined acids, which, if free HCl is present, means the combined HCl. The total secreted HCl is obtained by adding the total free HCl to the total combined HCl. If free HCl is absent from the stomach-contents, the combined HCl which may be present is estimated by Ewald's modification of Sjoqvist's method. Contrary to his previous statement, Hewes now declares that further investigation has convinced him that the use of dimethyl and alizarin alone, as indicators for free HCl and combined HCl, gives such erroneous results that it is not satisfactory even in clinical work.

A. E. Austin [1] describes a new method for the **determination of the digestive power** of the gastric juices. He first precipitates the albumin, and then precipitates the albumoses and peptones by phosphotungstic acid, and centrifugates in the Esbach tube. The results obtained by this method were compared with those furnished by Kjeldahl estimation, and Austin believes that his method has only a slight error. The amount of albumoses and peptone present varied considerably in different diseases investigated, which was probably owing, in Austin's opinion, to variations in the absorptive power.

A. Cohnheim and H. Krieger [2] describe a method of **determining the** amount of **combined HCl** present in the gastric juice. This consists in the addition of a salt of phosphotungstic acid to the gastric juice, thus precipitating the insoluble phosphotungstate of albumin. The most satisfactory method was the use of calcium phosphotungstate, which is made by adding calcium carbonate to a boiling 4% solution of phosphotungstic acid until the solution becomes neutral. The result of the addition of this salt is the precipitation of the phosphotungstate of albumin and a setting free of the HCl, which combines with the calcium; consequently the acidity decreases. They determined the amount of free HCl present and the total acidity. To another portion they add the phosphotungstate of calcium, filter, and then determine the acidity. The difference between this acidity and the total acidity shows the amount of combined HCl.

V. Harley [3] presents a general discussion on the diagnosis of diseases of the stomach by mechanical methods. There is nothing specially new in the descriptions of methods. He advises distention of the stomach with carbonic acid gas. [This method, in our experience, is far less satisfactory, and usually more distressing to the patient, than distention through a tube with air.] In cases in which there is an excess of HCl the amount of table salt and of spices should be reduced and stimulating substances should be used. When HCl is absent or deficient, the patient should partake freely of table salt. In cases of neurotic dyspepsia he has found internal faradism valuable; of drugs, strychnin is undoubtedly the most important in these cases. He advises that fluid should not be taken with the meals. [This is sometimes an inadvisable restriction, as some persons take little food, and suffer in nutrition unless they are allowed to wash it down with small amounts of fluid. In atonic cases, however, fluid should be restricted as much as possible.] Harley believes that what was formerly called catarrh of the stomach is often to be classed under the head of neurosis. [We must disagree with this, and state our conviction that most cases of gastric disturbance are due to more or less strongly marked organic alterations in the stomach, and not to pure neurosis.]

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[1] Boston M. and S. Jour., Mar. 8, 1900.

[2] Münch. med. Woch., Mar. 20, 1900.

[3] Practitioner, Oct., 1899.

Roth [1] has investigated the **secretion of pepsin** in gastric diseases by using the method of Mett, in which egg-albumen is coagulated in glass tubes of a diameter of 1 mm. or 2 mm., the coagulation being made uniform. These are placed in the filtered gastric juice. He decides that the variations in the secretion of pepsin are quite as great as those in the secretion of HCl. Hyperpepsia was found in cases of ulcer and of nervous disease, while in chronic gastritis, carcinoma, and nervous disturbances the amount of pepsin was frequently found to be small. Investigation of the amount of pepsin is more valuable in treatment than in diagnosis.

A. Baer [2] discusses the significance of the **yellow coloring-matter of the stomach-contents** often found upon adding alkalies. This peculiar lemon-yellow color was described some years ago by Spitzer. It is seen in the filtered contents when alkalies are added. Baer finds that its intensity is directly related to the presence of dextrorotatory substances in the stomach. When Trommer's test was positive, this color test was positive; while when the stomach-contents were levorotatory, the reaction was negative. He found that it occurred particularly after the use of carbohydrate foods, and he believes that it is dependent chiefly upon the nature of the food taken. It does not occur after exclusively proteid meals. He could not isolate the substance causing the reaction, but he believes that the reaction is of no special value, as it varies according to the character of the food, the nature of the gastric secretion, and the condition of the motor power of the stomach.

Friedberger [3] discusses the **condition of the urine** in diseases of the stomach. The more interesting part of the report relates to his investigation of the value of the determination of the amount of pepsin in the urine by Troller's method. He concurs with Troller in believing that the pepsin in the urine is in direct relation to the amount of gastric juice secreted, but it varies considerably even in normal persons. Its marked reduction is, however, of some value in diagnosis in cases of diminished secretion of gastric juice.

A. Jones [4] refers to the previous work which has attempted to show a definite **relation between the amount of HCl** in the stomach-contents and the presence or absence of **indican** in the urine. He records a series of 162 cases which show that there is no constant relation between any degree of HCl in the gastric contents and indicanuria. Of the cases, 85 showed free HCl and absence of indicanuria; 36 showed free HCl with indicanuria; 25 showed absence of indicanuria and absence of HCl; while in 16 there was absence of free HCl and indican was present in the urine. Jones reviews the recent work on indicanuria, and then directs attention to the fact that besides the condition of the free HCl, it is important to know the amount of combined chlorids and the degree of gastric digestion of proteids before we can have definite reasons to state that indicanuria will be present or absent. Indicanuria is certainly not dependent upon conditions of the stomach alone, but is largely influenced by the conditions in the intestine.

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[1] Zeit. f. klin. Med., Bd. XXXIX, p. 1.

[2] Berl. klin. Woch., Aug. 7, 1899.

[3] Deut. Arch. f. klin. Med., Feb. 6, 1900.

[4] N. Y. Med. Jour., April 28, 1900.

Leuk [1] contributes an interesting paper concerning the practical **value of diagnosis by means of fragments** of the gastric mucous membrane, first discussing the histology of the stomach and giving a series of measurements of the glands and connective tissue and of counts of the number of glands normally present in the mucous membrane at various points. He believes that the only useful sign of interstitial gastritis is increase of the breadth of the connective tissue; but if the connective tissue is much increased in some sections while the relations with the epithelium remain normal, he thinks that this is probably a mere temporary infiltration. While interstitial gastritis may be readily recognized in portions of tissues from the fundus in the pyloric region, the connective tissue is so irregularly distributed that it is very difficult to make a diagnosis from small fragments, even when removed at operation. He is very skeptical concerning the value of examination of fragments washed out from the stomach, since these fragments are so small. As to the changes in the number of glands, and a diagnosis from such changes, Leuk believes that a decided increase may be recognized in the region toward the pylorus; but this can not be done in the fundus, since the glands are so close together that the increase in number produces only a stretching of the mucous membrane, and under the microscope in small sections about the same number of glands to the field are seen. If an interstitial gastritis existed at the same time, there would be still greater confusion. The relative number of principal and parietal cells may vary so greatly normally that conclusions from observing them can be drawn only with great hesitancy and care. He does not believe that it is possible to diagnose carcinoma from atrophic gastritis by the examination of fragments unless actual carcinomatous growths are found, since other changes are almost exactly the same in the two diseases, and he has little faith in the diagnosis of carcinoma from the examination of fragments washed out from the stomach. [We have had occasion to express views somewhat similar to those of the author just quoted in the matter of diagnoses of gastric disease based upon microscopic examination of fragments of mucous membrane. The changes in the mucous membrane that may result from partial detachment and subsequent degeneration or digestion frequently prevent even an approximation to an accurate opinion.]

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[1] Zeit. f. klin. Med., Bd. XXXVII, Hefte 3 u. 4.

Fürbringer [1] advises the inflation of the stomach by introducing the tube part way into the esophagus, and then **inflating with the mouth.** He has previously advised the introduction of the tube completely into the stomach, with subsequent mouth inflation, but the result was often a mouthful of stomach-contents. This is avoided by the method which he now recommends.

C. D. Spivak [2] has used **auto-insufflation** of the stomach in distending this organ. He introduces just within the mouth of the patient a stomach-tube with a lateral opening. The patient then tightly closes his lips and blows, and this distends the stomach. He asserts that besides reducing the amount of apparatus necessary, it is easier for the patient to control the amount of air used and to avoid irritation of the stomach. [A Davidson syringe is usually easily procured and serves very well to inflate the stomach. The stomach-tube need not, therefore, be mutilated in the way the author suggests.]

C. S. Fischer [3] describes an **arrangement of gastric instruments** which he has devised in order to facilitate the examination and treatment of the stomach. There is an outer tube and 4 smaller ones, the object of the arrangement being to introduce with the smaller tubes an electrode, a douche, etc., without removing the larger tube in the different manipulations, hence saving distress to the patient and avoiding irritation of the esophagus. There are also special stop-cocks attached to the outer tube, and other details devised for the purpose of facilitating the work.

## GENERAL CONSIDERATIONS CONCERNING GASTRIC DISEASE.

A. Mathieu and Morichau-Beauchant [4] review the question of **perversions of appetite,** describing the **painful** and **nauseating** forms of abnormal hunger and a **form associated with great anxiety.** The last-named variety occurs especially in nervous subjects, though sometimes it has not the least relation to hysteria. When the persons become hungry, they are at the same time seized with a sense of the most profound anxiety. It is always wise for such persons to carry some food with them, as eating a small piece of bread or chocolate will usually protect them from these extremely unpleasant attacks. The painful form of hunger is most frequently noticed in persons with hyperchlorhydria, but it is occasionally found when the chemistry of the stomach-contents seems normal. Sometimes it is associated with a sense of nausea; at times nausea alone may be the most striking accompaniment of the hunger. Hunger accompanied by nausea is most frequently observed in young women, and is likely to come on late in the afternoon, the time when neuropathic subjects are most pronouncedly depressed in general; it is sometimes accompanied by marked salivation, and the saliva may be partly swallowed and then regurgitated from the esophagus. Sometimes no sense of hunger is noticed, but there is intense nausea, which disappears after taking a little food. These cases are not infrequently distinctly hysteric.

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[1] Deut. med. Woch., Oct. 5, 1899.

[2] Phila. Med. Jour., Feb. 3, 1900.

[3] N. Y. Med. Jour., Dec. 30, 1899.

[4] Gaz. des Hôp., Mar. 23, 1900.

F. F. Hewes [1] has carried on an investigation of 250 cases in order to determine the **relation of the subjective and objective signs of disease** of the stomach to each other. In 120 of the cases the stomach-contents were practically normal. The other cases showed hyperchlorhydria, hypochlorhydria, anachlorhydria, or hyperacidity due to organic acids. The result of his investigations was the conclusion that most symptoms were seen in all forms of disturbance; one symptom common to all cases was distress in the epigastrium. The intensity of the symptoms seemed to bear no relation to the objective signs; there may be marked hyperacidity with slight or severe symptoms, and with marked symptoms there may be little alteration in the gastric contents. He thinks, therefore, that the symptoms depend upon varying conditions of the stomach itself, this organ showing different grades of irritability. [The author's study bears out what general clinicians have long recognized, that too much stress has been placed upon chemical analyses of the stomach-contents. Clinical experience convinces us that the artificial classifications of gastric disease, based upon the gastric chemistry, are of little value.]

E. Bäcklin [2] has made a study of **regurgitation and its relation to other conditions.** In 338 cases which he studied he found that this symptom was present in 192. It had no constant relation to any form of gastric disease. It was more commonly absent than present in gastritis, however, but was more frequently present than absent in neuroses. It is extremely common with atony of the stomach. The fact that the regurgitation decreases as dilation of the stomach becomes more severe Bäcklin attributes to weakness of the pylorus, and he mentions a case to show that such weakness of the pylorus does occur, the evidences being chiefly the development of a constant inflow of bile into the stomach. In other cases it may be observed that the food is for a time retained for an excessively long period in the stomach, but ultimately, according to Bäcklin's belief, weakness of the pylorus causes premature expulsion from the stomach.

O. Rosenbach [3] discusses the importance of **diseases of the urinary apparatus,** both functional and organic, in **causing dyspeptic symptoms.** He presents a number of theoretic considerations, stating positively, without much ground therefor, that the secretions of the mucous membrane of the ureters, bladder, and urethra are of great importance in providing the elements of the urine and in maintaining normal conditions of metabolism. He therefore believes that any disturbance of the functions of the mucous membrane of the urinary tract is of marked importance to the general economy. A point of some interest is his insistence that the localized area of paralysis in the entire absence of true stenosis produces the same clinical results as are observed with mild stenosis, since the local paralysis interferes with the progress of the propulsive wave that follows along these canals, and therefore some stagnation results. Many minor lesions may cause paralysis, as local adhesions, small ulceration with thickening of the walls, and similar conditions. His description of the clinical picture in dyspepsia from motor insufficiency of the urinary apparatus is that it usually occurs in men in the sixth decad of life who have various dyspeptic signs, are likely to tire readily, frequently have chilly sensations, and often have subnormal temperature. They are obliged to pass urine frequently in the night and defecation is likely to be incomplete. The physical examination shows a palpable bladder, one that is likely to be distended very readily and is incompletely emptied. The urine shows no evidences of disease except excess of mucus and a few pus-cells. He believes that the cases should be treated by reducing the amount of fluid taken, in order to diminish the demand upon the motor power, by excluding irritating diet, by keeping the patients at rest, and by giving massage and faradization in the region of the bladder. Strychnin should be used in large doses. If there is paradoxic ischuria, catheterization should be carefully instituted. The symptoms caused by the disturbance of the urinary tract are apparently believed to be due to autointoxication. The prognosis is said to be good unless the cases are far advanced, in which event they are likely to die of uremia. [The paper is suggestive, and emphasizes the importance of noninflammatory disturbance of the urinary tract in causing digestive trouble, but it is marred by a great amount of speculation and by an unfortunate tendency to multiply new terms without limit.]

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[1] Boston M. and S. Jour., May 17, 1900.

[2] Arch. f. Verdauungs-Krankh., Bd. v, Heft 3.

[3] Deut. med. Woch., Aug. 17, 24, and 31, 1899.

Sigaud[1] discusses the **changes in the form and volume of the abdomen** and the clinical importance of the same, insisting that too little attention is given to inspection of the abdomen and to the observation of alterations in its form and volume. He describes two essential forms of change that may be seen, the first being an evidence of a feeble digestive tract, and showing itself as a flattening of the abdomen and a projection of the walls sidewise when the patient lies flat on the back, while when standing the suprapubic and iliac regions appear prominent and the epigastrium and lateral regions are abnormally flat. This is due to a ready relaxation of the walls of the stomach and intestines and a tendency to drop downward toward the pelvic cavity. The second class is represented by those who have what he terms "strong" digestive tracts with prominent abdomens. Such cases are very frequently seen in extremely young children; they present very prominent abdomens, and this condition lasts usually for years. Sigaud insists that one should look with care for the early alterations that show the beginning of loss of gastro-intestinal tone in such cases. These are: marked tendency to notable alteration in the size of the abdomen from day to day, a tendency for the abdomen to grow flatter and to become relaxed, and a frequent alternation of constipation and diarrhea. He considers these changes of serious importance in prognosis, as he thinks such subjects likely to have very grave gastro-intestinal abnormalities after the compensation is lost.

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[1] Rev. de méd., Sept. 10, 1899.

J. C. Roux [1] discusses the **tender point** frequently found in the center of the epigastrium in the middle line **in cases of gastric disease.** It is most marked in gastric ulcer, but he notes that it is frequently present in alcoholic gastritis and in other gastric disturbances. The chief question has been as to its cause, some authors attributing it to direct tenderness of the gastric ulcer, others considering it due to contracture of the pylorus. Neither explanation is satisfactory, however, as ulcer is not always present, is variable in situation, and the pylorus is very movable, while the pain is always at the same point. Roux considers that the point of pain corresponds to the ganglia of the solar plexus, and states that in making numerous necropsies of cases upon which this point of tenderness had been present shortly before death, and in which he had marked the point carefully with silver nitrate, he found that its situation was directly over the sympathetic ganglia. He describes an instrument which he has had made, and calls the gastric esthesiometer, its use being to determine accurately the degree of tenderness in the epigastrium by learning how much pressure can be applied to the epigastrium without causing pain. He finds that the tenderness varies at different times of the day, being least marked in the morning, more marked toward evening, and most marked after taking food. The effects of different foods also varied, those which remained longest in the stomach, as beef, causing more pain than others, such as eggs. Alcoholic drinks caused the tenderness to increase decidedly. Emotion, neurasthenia, and similar conditions caused it to increase, while it was diminished after the use of counterirritants over the epigastrium, as, for instance, blisters. Cocain decreased it, and in one instance of gastric ulcer the tenderness was found to decrease very greatly after a hematemesis. Roux considers this tenderness a direct indication of gastric irritability, and thinks that the determination of its intensity is of importance.

K. Faber [2] directs attention to the occurrence of a **reflex hyperesthesia of the skin in digestive diseases.** This is determined by picking up the skin between the thumb and finger and pressing it gently. No discomfort is experienced normally, but in certain cases of gastro-intestinal disturbance pain results. The areas over which this hyperesthesia may be observed were found to correspond quite closely with Head's description of the areas corresponding to spinal segments. There was some apparent relation between the location of the disease and the areas of skin affected. In disturbances of the stomach the areas were chiefly in the upper portion of the abdomen, while affections

[1] Rev. de méd., Nov. 10, 1899.

[2] Deut. Arch. f. klin. Med., Bd. LXV, Hefte 3 u. 4.

of the intestine caused the appearance of hyperesthesia over the lower portion of the abdomen. He reports 29 cases of hyperesthesia of the character described; in other words, about 15% of the cases of gastro-intestinal disturbance observed presented this sign. The cases were seen exclusively in women. In treatment Faber observed that while the hyperesthesia is likely to vanish with the cure of the gastro-intestinal trouble, in some cases it remained, and it was necessary to use local treatment to relieve the patients of their discomfort. For this purpose he found faradization, massage, and blisters most effective.

A. Robin and Leredde [1] insist again upon the importance of **disturbance of the stomach** in the **causation of affections of the skin.** They report 422 cases of dyspeptic disorder, in 129 of which there was functional or organic skin trouble. They consider that these can not be classed as mere coincidences. Acne was very common, eczema was seen 27 times, hyperhidrosis the same number of times, and furunculosis, urticaria, prurigo, and dermatitis were repeatedly seen. They insist upon the frequency with which eczema is caused by disturbance of digestion. The cause, they believe, is gastric fermentation. In these cases they have frequently determined the presence of organic acids in large amounts in the stomach-contents. They also noticed that the sweat was frequently exceedingly acid, and that when jaborandi was given the first portion of sweat secreted was very acid, and that subsequently this fluid became less acid. They have also determined the presence of lactic acid in the sweat in such cases. They believe that these facts support their contention that there is intoxication with organic acids. In the treatment, if there is lactic acid fermentation they recommend the fluorid of ammonia; if there is butyric acid fermentation, erythrol is useful. They also consider it important to improve the nutrition of the patient and to limit the tendency to excessive accumulation of fat.

Leredde [2] describes a case of **dermographism** in a dyspeptic subject without signs of hysteria. The gastric juice contained large quantities of butyric acid, and treatment directed to this condition caused the dermographism to disappear entirely.

E. Laborde [3] investigated the **influence of various alcohols** upon the digestion of albuminoid substances. He digested egg-albumen with pepsin or trypsin in the presence of various alcohols, and found that isobutyric alcohol, glycerin, and malic acid in the proportion of 5 : 1000 favored peptic digestion; methyl alcohol in very weak solutions favored digestion somewhat, while ethyl and propyl alcohols, lactic and tartaric acids, mannite, and glucose retarded it. Pancreatic digestion was slightly increased by methyl and isobutylic alcohols, glycerin, and glucose; it was somewhat decreased by ethyl and propyl alcohols, by malic, lactic, and tartaric acids, and by mannite.

R. Stern [4] divides the **results of injury to the mucous membrane** of the stomach from external violence into two forms. In the first healing occurs rapidly; in the second a chronic affection is produced. In the first group there is usually bloody vomiting, which may be repeated. It is associated with gastric disturbance of varying character. The symptoms commonly disappear in about 2 weeks, though they may last several months; they may closely resemble gastric ulcer. In the second group the condition becomes chronic and the prognosis is not very favorable. Stern does not believe that trauma can be considered of great importance in producing gastric carcinoma, but it is probable that it may accelerate the growth of an already existing tumor.

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[1] Bull. Acad. de méd., July 18, 1899.

[2] Gaz. hebdom. de méd. et de chir., Sept. 21, 1899.

[3] Compt. rend. de la Soc. de Biol., Oct. 21, 1899.

[4] Deut. med. Woch., Sept. 21, 1899.

## GASTRITIS.

Dieulafoy[1] describes cases of **hemorrhagic ulcerative gastritis** due to the **pneumococcus.** In 2 cases, both in men, pneumonia was followed by severe gastro-intestinal disturbances, with vomiting of blood and with signs of peritonitis. The postmortems in the two cases showed general pneumococcus infection; in one case the stomach showed hemorrhagic erosions which involved the mucosa, and in the other case there were hemorrhagic foci in the mucosa.

M. Einhorn[2] describes 4 cases in which **mycelia and spores of molds** were found in the washwater from the stomach. Three of the cases were hyperchlorhydria, and in one of these there was periodic gastrosuccorrhea; the fourth case was chronic gastritis with gastric erosions. The mold appeared macroscopically in the form of small blackish-gray and brownish-green flakes, which consisted almost entirely of the spores of the mycelia. The masses also contained some mucous corpuscles and epithelial cells; the presence of these was thought to indicate an intimate connection between colonies of the fungus and the surface of the mucous membrane, and to show that the fungus had actually grown upon the surface of the stomach. Such growth probably never takes place in the normal stomach, but the mold may lodge in a fold of the mucous membrane and from this point grow widely over the stomach. The relation which the mold bore to the causation of the symptoms was in much doubt, but probably the symptoms were, at any rate, made worse by its presence. In treatment Einhorn recommends irrigation of the fasting stomach, the use of the gastric douche, and the application of an antiseptic silver nitrate spray.

W. A. Bastedo[3] describes a case in a woman of 26 which began with severe symptoms of gastritis. In the vomit numerous particles were found which consisted almost entirely of **saccharomyces albicans,** and Bastedo believes that it was a case of gastritis caused by this fungus. The patient recovered completely.

R. C. Larribee[4] describes as **gastric tetany** the case of a woman of 26 who after dietetic indiscretion had headache and syncope, followed by spasmodic contractures of the arms, with slight paresthesia, the contractures lasting about 20 minutes. She improved after vomiting. Trousseau's symptom was present for a few days and then disappeared. The Chvostek symptom was absent.

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[1] Presse méd., Nov. 4, 1899.

[2] Med. Rec., June 16, 1900.

[3] Med. News, Mar. 24, 1900.

[4] Boston M. and S. Jour., Nov. 2, 1899.

M. Einhorn[1] describes 16 further cases of **erosion of the stomach.** The acidity varied from entire absence of HCl up to hyperchlorhydria. The signs of the affection were the presence of small pieces of gastric mucosa, a general feeling of weakness and languor, and discomfort or pain in the epigastrium shortly after taking food. There were numerous other symptoms, but none that were at all distinctive. In 2 cases the affection was associated with tuberculosis of the lungs; in one of these the gastric symptoms preceded the pulmonary by several months. This patient had achylia gastrica; but the pieces of mucosa showed the presence of glands. One patient was syphilitic; the occurrence of erosions in this case seemed to be coincident with a papular skin eruption, and it was thought possible that the erosions were the result of an eruption in the stomach. Specific treatment had no effect. Four cases were associated with chronic catarrh of the stomach, which condition is believed to have an important etiologic relation. The treatment was chiefly spraying with a solution of silver nitrate, 1 : 1000 or 2 : 1000, and the use of intragastric galvanization; and, if this was not successful, a long rest and a change of scene.

## FUNCTIONAL AND NEUROTIC DISTURBANCES.

P. Weber[2] reports what is apparently a well-authenticated case ot **hysteric vomiting of enemas.** The patient was a hysteric woman of 22 who had fecal vomiting, abdominal distention, and pronounced constipation. A demonstration of the existence of vomiting of intestinal contents was made by giving enemas of castor oil and methylene-blue. It was repeatedly observed that the oil and methylene-blue appeared in the vomit within 10 minutes after the introduction of the enema into the rectum. The suspicion that a fistulous communication existed between the stomach and the colon proved, upon laparotomy, to be unfounded. The patient finally recovered after a laparotomy by Treves, the mere operation seemingly having been active in producing recovery. Weber refers to the case of Jaccoud's in which autopsy after fecal vomiting showed the ileocecal valve to be perfectly normal. Weber suggests that the vomiting is produced by spasmodic contraction of the lower end of the bowel, which results in accumulation of the feces above this, and this causes reverse peristaltic action similar to that produced by complete occlusion of the bowel resulting from organic cause.

L. Schüler[3] insists, with others of the students of Strauss and with Strauss himself, that the **symptoms of hyperacidity** are not always accompanied by excessive acidity of the stomach-contents, and that excessive reaction to stimulus must be determined in some other way than by the mere investigation of the grade of acidity of the stomach-contents. Schüler gives an interesting report of some investigations which apparently show that in cases with the irritative symptoms which commonly indicate the presence of hyperacidity, whether the acidity be found high or not, one will always find a **blue or bluish color** upon testing **with iodin,** and the **specific gravity** will always be found low. The normal specific gravity is ordinarily from 1015 to 1020, while a specific gravity below 1010 is practically never found except in hyperacidity or hypersecretion. The reason that the acidity is not found elevated, and that the starch digestion is interfered with, is difficult to state, but it is possible that the stomach reacts unduly quickly, rapidly reaches a high point of acidity, and that the acid wave is descending already at the end of the hour when the stomach-contents are removed.

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[1] Arch. f. Verdauungs-Krankh., Bd. v, Heft 3.

[2] St. Bartholomew's Hosp. Rep., vol. XXXIV, p. 314.

[3] Deut. med. Woch., May 10, 1900.

A. N. Blodgett [1] describes a case of **cyclic vomiting** which occurred in a boy of 13 who was markedly precocious and had a neurotic family history. Examination of the case showed no abnormalities, but about once a month paroxysmal attacks of vomiting appeared, which lasted from two days to a week. Preceding these attacks the patient's general condition was depressed and he had some fever. During the attacks he vomited large quantities of gastric contents, which often contained food that had been taken days before and substances, such as wool and feathers, which the patient had swallowed in the attacks of voracious appetite that usually preceded the vomiting. During this preliminary stage of abnormal appetite the patient's moral character seemed perverted. The attacks were prevented by giving laxatives and emetics during the preliminary stage of excessive appetite.

## TREATMENT OF SECRETORY CHANGES.

W. Backmann [2] presents the results of experimental studies on the **dietetic treatment** of **hyperacidity.** He administered test-meals composed of beefsteak, eggs, and milk, with at times butter and cream, as examples of animal food; and other meals of bread, oatmeal, and potatoes as examples of vegetable food; and also made combinations of these. He made a series, in all, of about 1250 examinations on 12 patients with hyperacidity. One point which he insists upon as of importance in such a study is that the caloric value of each of the meals tried should be kept at about the same point in order that the results may actually indicate the value of the meals. The results were that free HCl appeared more quickly after taking the vegetable foods, but the animal foods caused a higher average value of HCl; butter and cream caused reduction in the amount of HCl; the HCl was low after milk, bread, potatoes, and oatmeal, higher after eggs, and highest of all after beefsteak. The peptic power was always about the same. The stomach was emptied most rapidly after the vegetable and milk meals, eggs were retained longer, and beefsteak longest of all; butter and cream caused a more protracted retention. Fats, however, evidently caused a retention in the secretion of HCl, and should be valuable in the treatment of hyperacidity. Beefsteak and eggs, especially the former, caused greatest irritation, and therefore seemed to be contraindicated, while the vegetable foods caused least irritation, and are therefore indicated, except potatoes, which increase the acidity through the production of lactic acid. There seems to be little disturbance of the starch digestion through the excessive acidity of the stomach.

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[1] Boston M. and S. Jour., Sept. 28, 1899.

[2] Arch. f. Verdauungs-Krankh., Oct. 28, 1899.

Simon [1] discusses the **influence of artificial sweating upon the secretion** of gastric juice. He notes that the sweat contains varying amounts of salts, and that the salts in the urine vary inversely as those in the sweat. It therefore seems probable that sweats might carry off the salts which provide the mineral portions of the gastric juice. Simon noticed by chance in one case that a sweat-bath reduced the acidity of the gastric juice greatly in a case of acid catarrh of the stomach. He therefore made systematic study of the effects of sweat-baths, and the results in 14 cases are recorded in this paper. The subjects were healthy persons, patients with hypersecretion, and others with nervous dyspnea, ulcer, and subacidity. As a rule, the sweat-baths caused marked reduction in the amount of free HCl in the gastric juice. The first effect of the bath was to cause a slight increase, but within 2 hours this was followed by a very marked reduction; there were, however, differences in the effect, and sometimes no reduction was observed. The most marked reduction was seen in normal persons and in those with hyperacidity. The amount of pepsin seemed to vary directly with the amount of free HCl. No distinct effect could be seen upon the motor power of the stomach. Sweats produced by pilocarpin had the same effect. Simon had used the sweat-bath treatment for the past 3 years in cases of hyperacidity due to ulcer or other causes, and has found it very useful.

M. P. Smithwick [2] reports cases of **hyperchlorhydria** treated with **silver nitrate** with successful results. He especially recommends that the stomach always be washed in order to remove the contents and to prevent the conversion of silver nitrate into silver chlorid; and that then lavage should be done with a 1 : 1000 silver nitrate solution, and the stomach afterward washed out with water.

Rosenbach [3] describes good results from the treatment of **nervous dyspepsia** with **chloral,** giving 2 or 3 grains of the diluted drug an hour or two after meals. If there is no organic disease he finds that the symptoms are much relieved by this treatment.

A. Théohari and E. Vayas [4] report the results of their investigations concerning the **influence of various drugs** upon the mucous membrane and upon the chemical secretions of the stomach. They introduced the drugs by the stomach-tube. Soda salicylate, potassium arseniate, and potassium iodid were each used for a period of about 6 weeks in therapeutic doses. Potassium iodid only was found to have any definite effect upon the mucous membrane; when the animals were killed, from about 2 to 5 hours after digestion had begun, they showed no traces of reticulum in or granulation of the cells. The HCl was diminished considerably in amount and the peptic power was lessened.

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[1] Zeit. f. klin. Med., Bd. XXXVIII, Hefte 1, 2, u. 3.

[2] Boston M. and S. Jour., Jan. 4, 1900.

[3] Therap. Monatsh., No. 9, 1899.

[4] Compt. rend. de la Soc. de Biol., Mar. 17, 1900.

Carter[1] recommends the use of **mercuric chlorid** in very small doses—$\frac{1}{2000}$ to $\frac{1}{1000}$ of a grain, given every hour for 10 or 12 hours of the day—in controlling vomiting and gastro-intestinal fermentation. Larger doses he considers may be harmful, and are unnecessary.

A. Mathieu,[2] in discussing the **use of hydrochloric acid** in diseases of the stomach, states that this may be used in large doses in the attempt to provide the amount of HCl that should be secreted by the stomach, or in small doses to excite the stomach to activity. The former method of employment had, in his hands, proved of little value; the latter had given him excellent results.

P. Le Gendre[3] reports 6 personal observations of the use of **animal gastric juice** in the treatment of severe gastro-intestinal disturbance. These cases showed marked gastric intolerance, vomiting, diarrhea, flatulence, and pronounced emaciation; they had hypopepsia and apepsia, and medication had been useless. The gastric juice of dogs rapidly produced good effects. In 2 cases the improvement lasted but a short time, but this was owing to the impossibility of continuing its use. The dose used was from 90 gm. to 150 gm. a day. Besides these personal observations, he described 3 favorable cases communicated to him by Sarrade. Analysis of the contents of the stomach in Le Gendre's cases showed that the animal gastric juice acted as a digestant itself, and also excited the secretory activity of the stomach.

Lannois[4] reports a case in which there had been progressive emaciation with marked disturbance of digestion and hypopepsia, which had resisted all treatment. He used **animal gastric juice,** and the patient recovered in 6 months and remained well at the time of the report. In discussion Barth mentioned the case of a woman of 25 who was of a nervous disposition, and who during pregnancy had an almost complete apepsia, with considerable emaciation. He used animal gastric juice, and, whether the result was due to suggestion or to the direct effect of the treatment, the woman rapidly improved and her digestion became normal.

T. Stevenson and A. P. Luff[5] have made experimental and clinical studies of somatose, and decide that it is a valuable nutriment, which is usually well borne, commonly improves digestion, and causes no disturbance of the gastro-intestinal tract. It also does not irritate the kidneys nor cause the appearance of albuminuria, albumosuria, or peptonuria, and during its use albuminuria usually disappears. The effect of somatose upon metabolism was found to be favorable.

Neumann[6] has made metabolic studies of soson, an albuminous product of meat and used as a substitute for meat. It is tasteless and causes no disturbance of digestion or of the general system, and Neumann believes that it may be used to replace the usual food-albumins. It is cheaper than meat.

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[1] Liverpool Med.-Chir. Jour., July, 1899.

[2] Gaz. hebdom. de méd. et de chir., Sept. 21, 1899, p. 1244.

[3] Gaz. des Hôp., Jan. 26, 1900.

[4] Gaz. des Hôp., Jan. 19, 1900.

[5] Lancet, Sept. 30, 1899.

[6] Münch. med Woch., Oct. 3, 1899.

## GASTRIC ULCER.

R. B. Greenough and E. P. Joslin[1] discuss the cases of gastric ulcer which were observed at the Massachusetts General Hospital between 1888 and 1898—187 in number. It occurred 5 times more frequently in women than in men, the average age of the men affected being 37; of the women, 27. Hemorrhage occurred in 81%. It caused death in only 1.27% of the women, but in 17% of the men. Perforation occurred in 3.2%, and always caused death. Of 114 cases, 80% were discharged cured or improved, but only 40% remained well after 5 years, and the mortality within this period from gastric disease was 40%; among the men it was 30%, among the women 9%. Ulcer is evidently more frequently fatal in men, and occurs later in life. It is also more frequently associated with hemorrhage. There seems, therefore, to be a difference in the ulcers in the two sexes. The fact that the mortality was 8%, and that 60% of the cases had no lasting cure, emphasizes the fact that surgical intervention should be undertaken in many cases even when it is not demanded by grave emergencies.

R. Saundby,[2] in a clinical lecture on ulcer of the stomach, states the proportion of women to men affected as 20 : 1. He considers anemia the most important factor in the causation. The mortality, he thinks, does not rise above 5%. In treatment he gives a mixture of magnesium sulphate, iron sulphate, and dilute sulphuric acid, and administers small amounts of milk and lime-water; if well borne, gradually increasing the quantity. If hematemesis has occurred recently he gives no food for 48 hours, administering instead nutrient enemas of 4 ounces of milk containing 1 egg and a teaspoonful of brandy.

Bèriel[3] reports an unusual case of **hour-glass contraction** of the stomach resulting from ulcer, in which another ulcer was present in the distal cavity of the stomach and caused a profuse hemorrhage, the blood being passed entirely *per rectum* without hematemesis. The woman died of tuberculosis of the lungs, and postmortem examination showed the condition mentioned.

J. H. Abram[4] reports the case of a girl of 27 who had previously had gastric symptoms which had been attributed to gastric ulcer, and who had an attack so closely resembling perforation of a gastric ulcer that she was removed to the hospital for the purpose of undergoing abdominal section. Her general condition was so grave that operation was postponed, and she died. The postmortem examination showed that there were no changes in the stomach, with the exception of a few small submucous hemorrhages. The only pathologic conditions described were a small amount of pus in the peritoneal cavity, enlargement of the spleen, and small gummas in the lung and liver. The spleen contained staphylococci, and the case was considered one of **staphylococcus infection.**

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[1] Am. Jour. Med. Sci., Aug., 1899.

[2] Brit. Med. Jour., Jan. 20, 1900.

[3] Lyon méd., Sept. 24, 1899.

[4] Liverpool Med.-Chir. Jour., Feb., 1900, p. 29.

M. Soupault,[1] in discussing the **treatment of gastric hemorrhage,** recommends the use of saline enemas to relieve the thirst and the administration of nutrient enemas. For the avoidance of diarrhea he uses a mixture of about 4 ounces each of thin bouillon and wine with two large spoonfuls of peptone. He believes that this, if properly administered, will pass the ileocecal valve and be digested in the small intestine, and that alimentation may be carried on in this way for several months. He notes, however, that it is extremely trying for the patient, and there is likely to be intense thirst and often great hunger. For the arrest of hemorrhage he recommends the injection of ice into the rectum or its application about the perineum for the reflex contractile effect. The application of heat, in the form of hot rectal enemas, is also useful.

## PERIGASTRITIS.

H. Westphalen and W. Fick[2] report 2 cases of **perigastritis adhæsiva pylorica.** The first patient had decided symptoms of obstruction of the pylorus, and was finally operated upon, the obstruction found, and pyloroplasty performed, with removal of some polypoid growths of the pyloric canal. The patient felt well for some time afterward, but then had severe pains, which were caused especially by shaking the body, by turning from one side to the other, and by similar procedures which might cause traction on adhesions. There was evidently no obstruction at the pylorus, as it could be determined by auscultation that fluids passed the pylorus readily. There was resistance in the pyloric region and pain when lying upon the left side; also, the patient had an epigastric hernia, and traction upon this caused pain in the region of the pylorus. Adhesions were diagnosed, the abdomen was opened, and the gastrocolic ligament was found fixed to the epigastric hernia. By this means the pylorus was constricted. Gastro-enterostomy was done, and the patient recovered completely. The second patient had symptoms of gastric ulcer. Treatment did not cause satisfactory improvement, and he developed signs of stenosis. He also had pain upon shaking the body, as on coughing; turning quickly from one side to the other caused pain, and there was reverse peristalsis. The most important symptom complained of, however, was marked pain in the pyloric region, which always came on most severely either a short time before or a short time after defecation. The diagnosis was stenosis of the pylorus as a result of ulcer; adhesions of the transverse colon. Operation showed that the diagnosis was correct, and gastro-enterostomy was performed. The authors divide the cases of perigastritis into those without disturbances of the motor power of the stomach, those which cause disturbance of the motor function, and those which are complicated by organic stenosis of the pylorus. Experience has shown that merely breaking up the adhesions is usually followed by a return of the symptoms; hence this operation is not advisable in most cases, but nothing more seems justified if operation is undertaken in the cases that show no disturbance of the motor power of the stomach, particularly if the adhesions are long strands which can easily be tied off. In the other classes of cases, however, nothing can be done with reasonable hope of success except pylorectomy or gastro-enterostomy, and the latter will usually be the operation chosen.

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[1] Rev. de Therap., No. 8, 1899.

[2] Deut. med. Woch., Dec. 28, 1899.

W. Calwell[1] describes what he calls the **adhesion type of dyspepsia,** the name explaining the character of the trouble. The characteristics which he points out are a history of gastric ulcer or other inflammatory disease about the pylorus, and local pain and tenderness in this region which is increased by exertion, sudden movement, or heavy meals, but decreases while at rest and when taking light, easily digested food. A bandage often gives comfort. The general health is usually good unless it be reduced from lack of food, since the patients are likely to be afraid to eat freely. Pain appears usually about a half hour after meals, being thus distinct from the pain of ulcer. The gastric juice shows normal conditions. In milder forms the diet should be regulated, gastric fermentation controlled, easily digested food given in small bulk, and some mechanical abdominal support ordered; but if improvement does not occur, and if the disorder is sufficient to interfere with the patient's occupation, or if he tends to become hypochondriacal, exploratory incision should be practised. This will usually reveal adhesions. Calwell believes that these adhesions often prevent the healing of ulcers, and sometimes cause severe gastric disturbances by involving branches of nerves. He finds that acute hemorrhagic pancreatitis is frequently associated with adhesions about the stomach.

W. S. Fenwick[2] gives a general discussion of **perigastric or subdiaphragmatic abscess,** based on the analysis of 56 cases, including 12 of his own, and of abdominal abscess resulting from perforation of a duodenal ulcer. The latter is rare, since only 22 cases were discovered, and 3 of these were personal cases of Fenwick's. Perigastric abscess practically always follows symptoms of gastric ulcer, which have usually been of prolonged duration. It is notable that hematemesis is a rare symptom preceding perigastric abscess; any preceding symptoms are often very slight. The onset is marked by the usual symptoms of perforation. The following course may be acute or chronic. This form is rapidly fatal; there is pain, dyspnea, frequently cough, retching and vomiting, irregular fever, sometimes chills, and usually within a few days secondary inflammation of the pleura or lung. This form usually lasts about 2 weeks. The chronic variety is characterized by the usual symptoms of onset, but ordinarily after this all signs subside or grow indefinite. There may be nothing noticed for a long time, or the patient may have ill-defined symptoms of abdominal abscess. Abscess following duodenal ulcer is rare, as stated. It occurs usually in females, the proportion to males being as 10 : 1. It commonly runs an acute course, and ordinarily terminates fatally. The physical signs differ somewhat from those of perigastric abscess in chronic cases, in that the mass is likely to form in the right hypochondrium or in the umbilical region, and in about 30 % it burrows down behind the peritoneum and presents in the right iliac fossa. There is pain in the right side of the abdomen and tenderness in the right hypochondrium and to the right of the navel, and often a mass appears in the hypochondrium or in the region above Poupart's ligament. The process may even travel across into the left groin or point alongside of the rectum. Complications of other forms of these abscesses are secondary abscesses, pylephlebitis, and rupture into the pleural cavity, into the pericardium, or into the peritoneum.

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[1] Brit. Med. Jour., Oct. 28, 1899.

[2] Edinb. Med. Jour., April, 1900.

## CHANGES IN FORM AND POSITION.

G. M. Ekwurzel [1] describes the case of a woman of 22 in whom autopsy disclosed a **vertical position of the stomach.** The first appearance was that of displaced colon, but further examination showed that the loop consisted of the stomach, practically vertically placed on the left side of the median line, and of the duodenum ascending almost vertically just to the right of the median line. The stomach and duodenum were closely connected throughout the length of the loop by areolar tissue and the gastrohepatic omentum. The transverse colon also formed a U-shaped loop, the lower border being near the symphysis.

H. W. Bettmann [2] gives a review of the literature upon **acute dilation of the stomach,** drawing attention to the fact that while chronic dilation is generally recognized, acute dilation has not received sufficient attention. He describes an interesting case in a girl of 17. After passing through an attack of typhoid fever she had in convalescence a sudden onset of abdominal pain and severe vomiting, with collapse, and extreme abdominal distention. Lavage brought forth a large quantity of thin greenish fluid and caused great improvement in the patient's condition. It was given repeatedly, and the distention decreased, the vomiting ceased, and the patient finally became entirely well after having almost died from the acute dilation of the stomach. It is a noteworthy fact that in most cases of acute dilation of the stomach there is persistent vomiting of greenish fluid, often in enormous quantities, and it has been suggested that this very free secretion of fluid may be the primary condition and may cause the dilation. Bettmann urges that it should be recognized that acute dilation is likely to occur in the convalescence or course of acute diseases. It should be treated by supportive measures, by the use of the stomach-tube as early as possible, and by rectal feeding.

T. B. Appel [3] records a case of acute dilation of the stomach which occurred in a boy of 17 after a severe injury in which there was a fracture of the femur, but apparently no injury to the trunk. The boy had severe pain in the abdomen on the day following his injury ; the pulse became rapid and the temperature rose somewhat. He had repeated epistaxis, and typhoid fever was diagnosed. After about 10 days he became much improved, however, but then had severe abdominal pain with copious vomiting, obstinate constipation, and increasing distention of the abdomen. He was thought to have intestinal obstruction, and was operated upon. The stomach was found greatly distended, but there was no obstruction. Death ensued and the autopsy showed only enormous dilation of the stomach. Appel considers it probable that the cause of dilation was paralysis of the stomach from the shock of injury, analogous to that which was observed at times in the intestine; and he thinks that it is probable that some of the cases of severe abdominal distention with paralysis of the intestine which follow surgical operation are due to injury of the sympathetic ganglia rather than to sepsis.

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[1] Phila. Med. Jour., Feb. 3, 1900.

[2] Phila. Med. Jour., Feb. 3, 1900.

[3] Phila. Med. Jour., Aug. 12, 1899.

W. H. Brown [1] records the case of a man of 55 who had what was thought to be intestinal obstruction. The attack had begun with severe vomiting and great pain in the abdomen, and the patient had collapsed. The abdomen was distended above the pubes, but was flat above the epigastrium and flanks; it was dull above the pubes and resonant elsewhere. Exploration of the fluctuating swelling with a needle withdrew a small quantity of dark green fluid resembling that seen in pancreatic cysts. Operation was performed, and a large thin-walled cyst was found and opened. The patient died. The necropsy showed that the supposed cyst was the stomach dilated to such an extent that it would hold about 5 pints of fluid. There was no obstruction of the pylorus and the other organs were healthy.

R. Kirch [2] reports a case of acute dilation of the stomach in a boy of 19. The lad had suffered from gastric disturbance for several days, and was taken suddenly with severe vomiting and distressing pain in the abdomen, and died in a few days. The postmortem examination showed marked dilation of the stomach, the greater curvature resting on the pelvic floor.

Richter [3] reports a case of **stenosis of the pylorus** in which sudden relief of the gastric symptoms and of constipation came on after the removal of 3 cherry-stones and 4 other fruit-stones during lavage. The man believed that he had carried these stones in his stomach for about a year, and Richter reports the case as being stenosis due to this cause. A tumor was palpable before the removal of the fruit-stones. The man recovered completely.

A. Hasenfeldt [4] describes the case of a child in which a stricture of the esophagus developed after swallowing lye, and the abdomen became very tympanitic. This was followed by effusion into the abdomen and by vomiting and diarrhea. Considerable emaciation ensued, but there was no pain or tenderness and no increase of peristalsis, and fever was absent. An operation was undertaken and showed extreme dilation of the stomach, due to **thickening of the pylorus,** the orifice being so greatly reduced in size that it would not admit a fine probe. Gastro-enterostomy was done, the esophageal stricture was afterward dilated, and the child practically became well.

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[1] Lancet, Oct. 14, 1899.

[2] Deut. med. Woch., Aug. 17, 1899.

[3] Deut. Arch. f. klin. Med., Aug. 18, 1899.

[4] Münch. med. Woch., Feb. 13, 1900.

P. Cohnheim [1] describes a case of **dilation of the stomach** with marked motor insufficiency which followed **traumatism.** There was at first evidence of the presence of ulcer; this was followed by pyloric contraction. The patient showed marked improvement under treatment, but disappeared from observation. It was afterward learned that he took to drinking linseed oil on the advice of friends, and almost entirely recovered. He stated that it was a common treatment in gastric disturbances among his own class of people. [Practically the same treatment has been used repeatedly in stricture of the esophagus, the preliminary use of oil making the passage of the sound much easier. It perhaps, as Cohnheim suggests, soothes the irritated mucous membranes, and in this way relieves spasm which has increased the stenosis.] Cohnheim admits that trauma may produce dilation of the stomach by first causing perigastritis or ulcer, but he attributes all acute dilations after trauma to obstruction of the duodenum from ileus. Chronic dilations resulting from traumatism are, he thinks, always secondary to the development of an ulcer or similar conditions. Chronic gastrosuccorrhea results from relative stenosis of the pylorus or duodenum in all cases. Dilation may result from pyloric spasm, and may even recover completely without operation, and many cases which are the result of organic obstruction may be partly relieved. He strongly disbelieves in the existence of dilation of the second degree (Boas) as a result of primary muscular weakness, and discusses the cases of severe dilation observed in Boas' clinic as illustrating this fact. Atony may go on to dilation if no obstruction develops. Atony and dilation differ in kind and not in degree only. One is the result of a general process; the other of a local obstruction.

J. H. Musser and J. D. Steele [2] report a **series of cases of dilation** of the stomach. They decide that the most valuable symptoms in determining the presence of atonic dilation of the stomach are the presence of fluid in a fasting stomach, the ready entrance of fluid into the tube with difficulty in the return flow, the absence of visible peristalsis, the evidences of fermentation and the concentration of the urine, and thirst. They consider inflation the most satisfactory method of determining the size of the stomach. The cases occurred in students, which led to the decision that this condition is not uncommon in students. Living in boarding-houses, carelessness in the use of alcohol and tobacco, and depression of the nervous system are the chief factors in causing the conditions in these subjects.

A. Albu [3] believes that the usual **method of dieting cases of dilation** of the stomach by giving them concentrated food and by limiting liquids is based upon false premises. It has been repeatedly shown that liquid foods remain a shorter time in the stomach than do concentrated foods, and Albu believes that the use of concentrated foods overtaxes the stomach more than liquids. He thinks that the only method of actually curing cases of stenosis of the pylorus or other instances of dilation of the stomach dependent upon mechanical hindrances is by operation, but he has been able to secure great improvement when operation was declined or was impossible by properly dieting the patients. He recommends a diet composed chiefly of milk, to which eggs and albuminous preparations, such as nutrose, are added. He also allows spinach, asparagus, and similar vegetables, as well as calf's-brain and sweetbreads. The milk is given not only pure, but in the form of various preparations, such as puddings. Albu gives small and frequently repeated meals, and also, because of the frequent feeling of fullness which these patients complain of, he introduces food through the tube after the morning and evening lavage. He has treated about 50 cases in this manner, with what he considers extremely satisfactory results.

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[1] Arch. f. Verdauungs-Krankh., Bd. v, p. 405.

[2] Am. Jour. Med. Sci., Feb., 1900.

[3] Deut. med. Woch., Mar. 15, 1900.

Lichty,[1] in discussing the **treatment of dilation** of the stomach, refers to it under the headings of hygienic, dietetic, physical, and therapeutic. Under the last-named heading the most important detail is the use of nux vomica or strychnin in atonic dilation. Constipation may demand the use of drugs, but this should rarely be undertaken, as the condition can usually be controlled by other measures. Diminution of HCl requires the use of acid, and excess may demand alkalies. The physical treatment includes massage of the abdomen, abdominal exercise, hot and cold douches, and electricity. Lichty believes that percutaneous application of electricity is as useful as intragastric. Under the hygienic treatment he urges that corsets should be abandoned, and that the clothing should be so arranged that the weight may fall from the shoulders and not upon the hips. His dietetic treatment differs from the usual recommendations, but approaches to that of Albu. He gives 2 glasses of milk with 2 raw eggs at about the time of the three usual daily meals, and in the middle of the morning and afternoon and at 9 P. M. he gives 2 glasses of milk. The patient should rest on the back or on the right side for half an hour after taking food in order to relieve the greater curvature of the stomach from pressure. Solid food is instituted when the weight has returned to about normal, and the diet is gradually increased.

M. Einhorn,[2] in discussing the **indications for operative treatment** of benign stenosis of the pylorus, states that any surgical measures should be postponed until it has been determined that medicinal treatment is insufficient and that there is still marked ischochymia. The mortality is practically 25%, even with good surgeons, and therefore operation should be delayed as long as possible in benign cases. In malignant disease, however, operative treatment is the only rational measure, and should be undertaken as soon as the diagnosis is made.

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[1] Med. Press, Nos. 31 and 34, 1899.

[2] Med. News, Nov. 25, 1899.

## CARCINOMA OF THE STOMACH.

W. Osler and T. McCrae[1] report a series of 7 cases of **latent carcinoma** of the stomach. They describe 3 kinds of cases. In the first class, of which a large number occur in almshouses and asylums, there are symptoms only of gradual enfeeblement, without any signs of local disease. In the second there is evidence of other disease which seems sufficient to account for the condition, and gastric symptoms are absent. In the third there are metastases which completely mask the existence of the primary disease. As instances of the second class they record 2 cases of nephritis in which gastric symptoms were absent during life, but autopsy showed carcinoma, involving in the one case almost the whole stomach and extending on to the esophagus, and in the other case also involving a portion of the esophagus. In another case there were signs solely of chronic phthisis, and pneumothorax was present; autopsy disclosed the presence of carcinoma of the stomach with secondary growths in the liver and lymph-glands. There was also tuberculosis of the intestines as well as of the lungs. A fourth case showed a remarkable number of thrombi of the superficial cutaneous veins, with marked and progressive anemia, the red corpuscles being reduced as low as 1,716,000. Nucleated red corpuscles were absent; the white cells were 29,000. Autopsy showed carcinoma of the pylorus. There are 3 cases reported in which the symptoms seemed largely attributable to secondary growths, and the signs of primary carcinoma of the stomach were practically entirely absent. In one of these cases exploratory operation for ascites led to the recognition of malignant disease of the stomach. The same authors,[2] in the discussion of **carcinoma of the stomach in the young,** review the literature of this question. The cases reported in children under 10 are only 6 in number, and all of them are subject to criticism concerning their postnatal origin. In the second decad there are 13 cases reported, while in the third decad there are a good many more. In the 150 cases of cancer of the stomach in the Johns Hopkins Hospital, 6—that is, 4%—were in patients in the third decad of life. The diagnosis in the first case was not confirmed, but was made because of the suddenness of the onset, the absence of HCl, the presence of lactic acid, and severe anemia with the general symptoms of cancer. In the second case there was a tumor of the stomach with secondary growth in the umbilicus; in the third case the general symptoms and local signs pointed to carcinoma, and operation showed a carcinomatous mass on the anterior curvature. This man died within 4 months after the symptoms appeared. In the fourth case the duration was only about 6 months. In the fifth case the duration was only about 4 months. The case was investigated postmortem, and histologic examination of the growths showed it to be typically carcinomatous. The sixth case had a history of having been twice in the hospital for acute gastritis, but had been free from gastric symptoms meanwhile. He presented the usual appearance of carcinoma and died after 5 or 6 months' illness. Five of the 6 patients had decided pain, and the same number had vomiting. Tumor was found in 5 of the cases, and in 1 of these only at autopsy. Mathieu has stated that fever is generally absent in such cases in the young, but 3 of these cases had fever, and a fourth gave a history of fever. Mathieu stated that the appetite is not usually notably affected, while Brinton has made a contrary statement. In the cases here reported the appetite was unaffected in 3, was lost in 2, and in 1 case it was not noted. The features of the cases that were most prominent are those that were previously noted by Mathieu; they were of remarkably rapid onset, and of extremely acute course.

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[1] Phila. Med. Jour., Feb. 3, 1900.

[2] N. Y. Med. Jour., April 21, 1900.

Osler and McCrae[1] also discuss **the blood in carcinoma of the stomach.** They report a series of examinations in 150 cases. The conditions found were usually those of secondary anemia. When the blood count is high or normal, there is usually obstinate vomiting or constant secretion of fluid in the stomach. The blood count may be found high even with great emaciation, and 3 cases are reported in which the count was over 6,000,000. The conclusions arrived at are that if the red cells are reduced below 1,000,000, this is strongly in favor of a diagnosis of pernicious anemia; and if megaloblasts are present, this also speaks for pernicious anemia, as they are rare in cancer. The number of the leukocytes or of the various forms of leukocytes is not of value in making a diagnosis. There is no special change seen in cancer. Digestion leukocytosis may be present or absent. It was seen in 10 of 22 cases investigated concerning this point. The leukocytes were decreased in nearly half the cases, while on the average they were found between 5000 and 20,000. The differential count in 22 cases showed an average of 81% polymorphonuclears, 9% small mononuclears, 8.5% large mononuclears and transitional, and 1.5% eosinophiles. The cases with fever showed a much larger average of leukocytes than those that had no fever. The presence or absence of ulceration or of metastasis had little influence upon the number of leukocytes.

A. Richter,[2] in discussing the secretion of hydrochloric acid, especially in carcinoma of the stomach, reports 2 cases of carcinoma, in both of which **free HCl was present** late in the disease, in one case in excessive amounts. Both patients had had nervous hyperacidity of the stomach for a long period, but carcinoma was diagnosed. From the observation of these cases, and because hyperacidity is so common in carcinoma developing on the basis of ulcer, Richter believes that in the cases in which HCl is present in carcinoma in excessive or normal amounts the cause is nervous excitability, resulting from irritability of the nerves or from the presence of an irritating ulcer. He does not assent to the teaching that secondary gastritis causes the reduction or the absence of HCl in carcinoma, as many cases of benign stenosis show severe grades of gastritis, but are accompanied by the excretion of large amounts of HCl. He believes that the loss of the secretion is generally the result of general weakness, blood changes, and impoverished nutrition of the nerves. Gastritis causes loss of HCl only when it is of the atrophic form.

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[1] N. Y. Med. Jour., May 19, 1900.

[2] Arch. f. Verdauungs-Krankh., Oct. 28, 1899.

A. Stolz[1] describes a case of **carcinoma** of the pylorus **with continuous gastrosuccorrhea** and with marked diminution in the secretion of HCl. The clinical appearance was that of carcinomatous stricture with dilation, the stomach always containing acid fluid in the morning even when food was absent. The acidity was due almost entirely to organic acid, though there was some combined HCl. Pepsin was present. Hence the fluid seemed to be gastric juice. The chlorids were secreted in large amounts even though HCl was very much reduced. After gastro-enterostomy the acidity was much reduced, because of the reduction in the organic acids, while the chlorids and the HCl showed but little change.

R. Seggel[2] describes 3 cases of carcinoma of the stomach which **involved the anterior abdominal wall.** In 1 case the external growth had followed along the tract of incision after gastro-enterostomy. In another case the growth in the abdominal wall, together with a considerable part of the stomach and some enlarged glands, was removed by operation, and a good recovery followed.

L. M. Bonnet[3] exhibited to the National Medical Society of Lyons the organs of a patient who during life had presented a tumor of the epigastrium of apparently cancerous nature. In the latter part of life he had diarrhea following almost immediately upon eating, and it was easy to demonstrate the presence of food in the stools when the food had been taken only a short time before. Hence a diagnosis of **gastrocolic fistula** was made, and autopsy showed the correctness of the diagnosis.

H. Strauss[4] describes a case of carcinoma of the stomach, complicated by subdiaphragmatic abscess, in which it was noticed during life that the stomach-contents always showed large amounts of pus. The gastric motility was good, but hydrochloric acid was absent. The ethereal sulphates of the urine were largely increased and there was albumosuria. The presence of a large amount of pus during the latter part of life caused a subphrenic abscess to be suspected. Strauss believes that if one finds considerable quantities of **pus in the gastric contents,** it is a **valuable sign** of carcinoma. If the amount is very large, however, it is likely to indicate the presence of an abscess communicating with the stomach. Repeated presence of blood in the stomach-contents is also an indication of carcinoma. Strauss has never seen a noteworthy amount of pus in the stomach-contents except in the case reported in this paper, and in 4 others of ulcerating carcinoma. In cases in which the motility is much disturbed it is often difficult to discover the pus because of the mass of stagnating food with which it is mixed. He also notes that he has discovered metastasis in the mediastinal lymph-glands, in Douglas' pouch, and in other situations by means of the x-rays, and that this has helped to clear up the diagnosis.

M. Soupault and M. Labbé,[5] in a pathologic and clinical discussion of cancer of the lymphatic glands, express their belief that too much emphasis has been laid upon the frequency of cancerous **involvement of the glands about the clavicle and in the inguinal region** in abdominal cancers. In 6 cases of cancer of the stomach they saw cancerous enlargement of the glands about the clavicle but once, and then there was generalized enlargement of the lymphatic glands. In 10 cases of abdominal cancer they found enlargement of the inguinal glands but once, and in this instance there was one small cancerous nodule on one side. They admit that enlargement of the glands in the inguinal regions is not infrequent, but in 2 cases of cancer of the stomach they found that the enlargement of the supraclavicular glands which was present was tuberculous, and they point out the undoubted fact that a dependence upon this sign may lead to diagnostic errors, as there are numerous other sources of glandular enlargement in this region, particularly infection from the organs of the chest or from the bucco-pharyngeal cavity. [Our experience coincides with the view of these authors. It is a common teaching that the supraclavicular glands are enlarged in cancer of the stomach, but this seems to be the result of handing down a statement from one writer to another. The inguinal glands undoubtedly show slight enlargement frequently, but this is often seen in persons without malignant growth, and can not be depended upon as a definite sign. We have frequently seen small nodules in the subcutaneous tissues of the abdomen in abdominal cancer, and they seemed to be of considerable diagnostic importance.]

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[1] Zeit. f. klin. Med., Bd. XXXVII, Hefte 3 u. 4.

[2] Münch. med. Woch., Dec. 5, 1899.

[3] Gaz. hebdom. de méd. et de chir., Mar. 8, 1900.

[4] Berl. klin. Woch., Oct. 2, 1899.

[5] Rev. de méd., Jan. and Feb., 1900.

J. C. Hemmeter,[1] in discussing the **early diagnosis** of carcinoma of the stomach, states that statistics seem to indicate that this disease is increasing in frequency. Hemmeter insists that cancer of the stomach frequently occurs before middle life, a fact that is usually overlooked. The bowels are usually abnormal, and show either constipation or diarrhea. The points of diagnostic value are chiefly the marked degree of emaciation and cachexia, even in relatively early stages, and the presence of lactic acid and of the Oppler-Boas bacillus. He has found lactic acid in 82% of cases, and the Oppler-Boas bacillus in 42 of 52 cases. He considers exploratory laparotomy advisable if the medicinal treatment of suspicious cases does not give rise to improvement within 3 weeks. Hemmeter [2] later directs attention to the occurrence of **pathologic mitoses** as evidence of malignant growth. He believes that the diagnosis may be made by examining fragments of the mucous membrane and observing the presence of pathologic mitoses, particularly if with this change one sees atypical formation of tubules.

## SYPHILIS OF THE STOMACH.

M. Einhorn [3] contributes an important paper on syphilis of the stomach, and divides specific disease of this organ into 3 classes: **gastric ulcer** of syphilitic origin, **syphilitic tumor** of the stomach, and

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[1] Med. Rec., Oct. 21, 1899.

[2] Phila. Med. Jour., Feb. 3, 1900.

[3] Phila. Med. Jour., Feb. 3, 1900.

**syphilitic stenosis** of the pylorus. He describes 2 cases in which there were convincing signs of gastric ulcer, but a history of syphilis led to the use of specific treatment, and the patients recovered practically entirely, as well as rapidly. In the second group he describes 2 cases in which there was a mass, apparently of the stomach, which, except for a suspicion of syphilis, would have been considered carcinoma. The first patient became entirely well after the use of specific treatment, and the mass completely disappeared. In the second case the mass disappeared under this treatment, though it subsequently returned. The man was put upon treatment again, but disappeared from observation. Two cases are reported in which there were signs of stenosis of the pylorus, and in one case a tumor could be felt in this region. One man improved greatly and the other apparently got well. Einhorn insists that the possibility of a syphilitic origin of chronic gastric trouble should never be forgotten, and we should recognize the fact that syphilis of the stomach is not so rare as past teaching would lead one to think.

J. W. Dalgliesh [1] describes 3 cases of syphilis of the stomach. The first occurred in a woman of 55 who had had severe epigastric pain after taking food, and marked hematemesis, together with decided emaciation. The patient did not improve upon the customary treatment for ulcer and she had a history of syphilis, so that treatment with iodids was instituted; this caused rapid improvement, and all symptoms disappeared. The second case exhibited epigastric pain with hyperacidity and marked general depression. The customary measures were ineffectual in this case, and antisyphilitic treatment brought about recovery within a month. In the third case a malignant growth had been diagnosed by several who had come in contact with the case, but the use of iodids caused the disappearance of the tumor and complete recovery of health.

## TUBERCULOSIS OF THE STOMACH.

M. Simmonds [2] criticizes Petruschky's statement that the use of tuberculin may demonstrate that tuberculous ulcers of the stomach are not infrequent. In 2000 autopsies Simmonds found but 8 cases of tuberculosis of the stomach, and one can not consider the results of tuberculin injection to be more important than pathologic research. Simmonds thinks that tubercular ulceration may be the result of abnormalities of the gastric juice. In one case he found a pyloric cancer and multiple small tuberculous ulcers. As a rule, tuberculous ulcers of the stomach give rise to no clinical symptoms, and the diagnosis is practically out of the question. If signs of ulcer occur in phthisical patients, one should suspect simple ulcer rather than tuberculous ulcer.

## FOREIGN BODY IN THE STOMACH.

F. Schopf [3] describes the case of a girl of 12 who had experienced marked epigastric discomfort, and had a mass in the epigastrium. It was considered to be a tumor of the stomach, but examination showed that it was a foreign body in the stomach, and incision disclosed a large mass of hair. The cause was the customary one: the child had been given to chewing her own hair, and also ate the hair of a pet dog.

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[1] Lancet, Aug. 12, 1899.

[2] Münch. med. Woch., Mar. 6, 1900.

[3] Wien. klin. Woch., Nov. 16, 1899.

# DISEASES OF THE INTESTINES.

## THE EXAMINATION OF THE FECES.

R. Schutz,[1] in writing of constipation and diarrhea, insists upon the value of examination of the feces. The most important result that may be attained is the determination of the **presence or absence of mucus,** practice enabling one readily to determine whether the quantity is pathologic or not. If pathologic quantities are present, in the absence of membranous colitis, the discovery indicates intestinal catarrh, except when there is marked acute diarrhea. With simple acute dyspeptic diarrhea there may be an excess of mucus; but if this persists after the diarrhea has ceased, it indicates catarrh of the intestine. The localization of the catarrh is determined after the method of Nothnagel. The presence of mucus without feces, or of scybala enveloped in mucus, indicates catarrh of the lower part of the large intestine. If the mucus is intimately mixed with the feces, it means catarrh of the upper portion of the colon or of the small intestine. The presence of mucus with numerous food remnants indicates disease of the small intestine. Nothnagel's yellow mucous grains and hyaline mucous particles are of little value in localizing the trouble.

A. Schmidt[2] discusses the **clinical importance of bits of meat** in the stools. After a series of examinations he has reached the conclusion that ordinarily these fragments are chiefly connective tissue and fat. Artificial gastric juice digests connective tissue much more rapidly than muscle tissue, hence the presence of these bits of connective tissue indicates disturbance of the stomach, since trypsin digests the muscle-fibers more rapidly. He also gave to a number of patients 100 gm. of well-chopped meat, and from his investigations decided that if connective tissue remnants are found, they indicate disturbance of the gastric functions; if muscle masses are found, they indicate disturbance of the intestinal digestion. Also, the presence of nuclei in the tissue remnants indicates disturbance of the intestinal digestion, since when intestinal digestion is normal, nucleins are digested, while gastric juice does not affect them. The nature of the disturbance of the stomach is not made evident by this method.

K. MacLeod[3] discusses the process of **"washing" dysenteric stools,** which is constantly carried out in India. The technic is to receive the stool into a vessel of considerable size, and then to pour water upon it from a height of a foot or two. If the masses are hard, they are broken up with a stick and allowed to settle, and the fluid is decanted.

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[1] Berl. klin. Wọch., July 10, 1899.
[2] Deut. med. Woch., Dec. 7, 1899.
[3] Edinb. Med. Jour., April, 1900.

The feculent matter floats and passes off with the fluid, pathologic products and heavy particles of feces subsiding. By repeated washings of this kind the material may be freed of offensive and unimportant matters and then readily examined. During the process and in the subsequent examination one may readily notice the presence of blood, the character of feculent masses, the state of digestion and of the excreta, often the character of the ingesta, the presence of mucus and its characteristics, possible sloughs from the intestinal mucous membrane, and the presence or absence of bacteria or amebas. He states that in this way one can also get a better idea of the progress of the case than by any other method.

A. H. Carter and C. A. MacMunn [1] describe 3 cases in which they observed a **peculiar pigmentation of the stools,** which were of a red color, resembling blood. The first case was seen in an old man who had chronic disease of the heart and cystitis, with intoxication and fever. The second was observed in a case of typhoid fever, and the third in acute pneumonia. At first there were streaks of red upon the surface of the stools, but after exposure and agitation the whole mass became blood-red. A chemical examination led to the conclusion that blood was absent, as were bilirubin and biliverdin, and that the pigment was closely related to stercobilin, though not identical with it. The feces contained a chromogen, which upon exposure to the air became converted into this pigment.

## DIARRHEAL AFFECTIONS.

I. B. Yeo [2] reports a case of **chronic diarrhea** which recurred at intervals for 3 years, and which was entirely resistant to the usual treatment. The patient had finally become gravely ill. The stools were very white, and for this reason pancreatic emulsion was ordered and the man was given milk diet. Improvement began, and continued rapidly, and there was no return of the diarrhea except when the pancreatic emulsion was stopped. [Many cases of this kind are instances of achylia gastrica, and the fact that pancreatic emulsion was so useful in this case suggests that it might have been of the same character.]

L. Brunton,[3] in a clinical lecture upon **sprue and Indian Hill diarrhea,** states that the distinction between the two is that in sprue there is distressing soreness of the mouth and tongue and of the anus, while this is absent in the other affection. In both there is the characteristic diarrhea with white, frothy, liquid stools and loss of nutrition and strength, which, unless the patient is well treated, progresses finally to such a degree as to cause death. The essential treatment is careful diet, which should, if possible, be absolute milk diet. If this can not be used, it is best to use absolute meat-juice diet. The most effectual medication seems to be bismuth with cannabis indica. Pathologically, the disease consists of aphthous ulcerations of the mouth, with denudation of the epithelium of the mouth, esophagus, and intestine, the epithelium being almost entirely lost and the mucous membrane very greatly thinned.

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[1] Lancet, Nov. 25, 1899.

[2] Lancet, July 22, 1899.

[3] Edinb. Med. Jour., Feb., 1900.

H. Solomon [1] describes a case of **infusorial diarrhea.** The movements were frequent, and there was considerable loss of weight and of general health. Examination showed round, shining, pear-shaped bodies with flagella, which were from 1 mm. to 18 mm. in length. There were also some round or oval bodies with a slightly granular protoplasm similar to the others, and some very small forms, which were exceedingly motile. The organisms live for a number of days in the cold. Cultures and inoculation experiments were negative. A sister-in-law of the patient had diarrhea with similar parasites in the stools.

L. Gaillard and R. Monod [2] report a case of **cholera nostras** which ended fatally. The bacteriologic examination of the stools showed the presence of only 2 micro-organisms, one of them the colon bacillus, and the other a diplococcus which proved to be the **enterococcus of Thiercelin.** The latter organism was believed to have caused the attack. Both organisms were found in the blood of the liver, spleen, and kidneys, while the diplococcus was found in pure culture in the heart blood.

## DUODENAL ULCER.

R. S. Thomas [3] describes a case of probable duodenal ulcer in a man of 24, in which there had been severe and rapid hematemesis and loss of blood from the bowel, together with pain in the right side of the epigastrium, which came on about an hour after meals. In one attack of hematemesis the man nearly died from loss of blood. An intravenous injection of saline solution caused rapid improvement, and he soon was out of danger, and recovered entirely. It is suggested that the duodenal ulcer might have been the result of **septic embolism** from a poisoned wound of the thumb from which the man had been suffering for some time when the symptoms of duodenal ulcer appeared.

## DYSENTERY.

Marchoux [4] describes an **epidemic of dysentery** which occurred in Senegal during the hot months. In all, 47 cases were admitted to the hospital, 2 of them proving fatal. The feces contained **large numbers of amebas.** The injection of small portions of the feces into cats *per rectum* resulted in the production of dysentery. Great numbers of amebas were found in the stools of the animals. These inoculations could be carried out in series from cat to cat, the series being carried to the nineteenth degree and the amebas being found throughout the whole series. Abscess of the liver was frequently found in the cat and amebas were present in the abscess. If the feces were heated to 45° C. for 35 minutes, rectal injection produced no disease in the cats.

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[1] Berl. klin. Woch., Nov. 13, 1899.

[2] Gaz. des Hôp., April 6, 1900.

[3] Brit. Med. Jour., May 5, 1900.

[4] Compt. rend. de la Soc. de Biol., Nov. 11, 1899.

S. Eldridge[1] gives an interesting account of an **epidemic of dysentery** which has been observed **in Japan for 20 years.** It began in the south, and gradually spread through almost the whole of the Japanese empire, the mortality being extremely high. The total number of cases is said to have been 1,136,096 ; and of these, 275,308 died, the average mortality being about 24 %. There has been but little success resulting from the attempts to control the epidemic, the striking lack of hygiene making it difficult to control such a disease. The people often throw dejecta into streams, and then use the water for drinking purposes. The disease is an acute catarrhal and ulcerative process situated in the colon and rectum. Eldridge refers to Shiga's results from his bacteriologic study ; he believes that he has found the specific cause in a short bacillus resembling that of typhoid fever. Shiga also used an antitoxic serum that reduced the mortality—which at that time was 37 %—to 8 %.

W. J. Buchanan[2] reports a series of **555 consecutive cases** of dysentery with only 6 deaths. The treatment was salines in most of the cases, **sodium sulphate** being used instead of magnesium sulphate. He prepared a 4-ounce mixture containing 1 ounce of the sodium sulphate, and gave ½ of an ounce of the mixture 4 times a day.

F. A. Rouget[3] reports upon his experience with **dysentery in Mauritius,** and states that it is not a very fatal affection if treated soon after the onset. The mortality in the prison hospital, where treatment is begun early, never goes above 8 %. The best results are obtained by the use of small doses of magnesium sulphate, which should be continued for some days after the stools have lost their dysenteric character.

H. J. Jervis[4] reports the cure of 11 cases of acute tropical dysentery in from 5 to 8 days with the magnesium sulphate treatment.

R. Schultz,[5] in discussing membranous catarrh of the large intestine and **mucous colitis,** states that he considers the latter an independent affection which differs from catarrh of the bowel. The chief distinguishing point is that in mucous colitis the secretion of mucus is periodic and the stools in the interval appear normal. If there is mucus in the stools in the interval, it must be considered that there is catarrh of the bowel. [The exact nature of mucous colitis does not appear from this description. Catarrhal inflammation may without doubt be paroxysmal.]

S. Keith[6] recommends the treatment of **appendicitis** by the baths at Plombières, the patient to lie for from 40 to 50 minutes covered to the neck with water at a temperature between 34° C. and 36° C., a spray of warmer water from a rubber tube about an inch in diameter being allowed to play over the appendix during the latter part of the bath. Some extremely good results from such a treatment are reported.

C. Georg, Jr.,[7] discusses the occurrence of **intestinal sand,** and describes a case in which there were attacks of colicky pain, pain in the back and general nervousness, and some enteroptosis. The stools contained a considerable quantity of sandy material, which when investigated was found to be composed of calcium, magnesium, and ammonium in small amounts, with some silica; microscopic examination showed that the sand was chiefly sclerenchyma. The origin of the sclerenchyma was found to be in gooseberry jam which the patient frequently ate, the same sclerenchyma being found in the cuticle of the fresh gooseberry. Regulation of the diet caused the sand to disappear from the stools, but the symptoms in the case persisted to a certain degree.

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[1] Pub. Health Rep., Jan. 5, 1900.

[2] Brit. Med. Jour., Feb. 1, 1900.

[3] Brit. Med. Jour., Nov. 18, 1899.

[4] Brit. Med. Jour., Nov. 25, 1899.

[5] Münch. med. Woch., April 24, 1900.

[6] Lancet, Mar. 3, 1900.

[7] Physician and Surgeon, May, 1900.

E. Schreiber [1] reports the case of a boy of 9 who had repeatedly had attacks of severe constipation with vomiting, the vomit being often fecal. When admitted to the hospital he was vomiting freely, his abdomen was somewhat distended but not tender, and he was obstinately constipated; the vomiting was not fecal, but was very severe. The vomit contained no HCl. He died suddenly, and there was found an old **torsion of the root of the mesentery** which had probably existed for years, and seemed to have been the cause of the previous attacks of temporary obstruction. This was produced by the duodenum taking a winding course beneath the ascending colon, so that the colon came to lie twisted about the root of the mesentery of the duodenum. The final cause of death had been a volvulus produced by the twisting of the jejunum and ascending colon. The ascending colon and the cecum were not attached to the lateral walls of the abdomen, but lay free, with a mesentery similar to that of the small intestine. Schreiber reports another case, in which there were attacks of extremely severe colic and rapid loss of strength with prostration, but without any fecal vomiting. This patient proved to have torsion of the mesentery with obstruction of the bowel, and Schreiber believes that if there is obstinate constipation with vomiting, even though the vomit is not feculent, and that if these symptoms are accompanied by a striking loss of strength, one should always consider the possibility of intestinal obstruction. He advises a laparotomy in such cases unless treatment causes almost immediate improvement.

W. N. Dunlop [2] discusses **thrombosis of the mesenteric vessels.** He states that the symptoms are acute if the arteries are thrombosed, while when the veins are affected the symptoms are usually more chronic. The cases are often mistaken for intestinal obstruction. In that described a man of 51 had severe pain in the hypogastrium and vomiting, with a flaccid abdominal wall. Blood was passed by the rectum, and the patient died. The autopsy showed pronounced congestion of the ileum, with thrombosis of the veins of this area. There was no evident cause.

C. Schlatter [3] gives the report of a case of **intestinal injury** in a man of 23 in which he resected a large portion of the ileum. The length, after the bowel had contracted considerably, was 192 cm. It has frequently been said that as much as 2 m. of the ileum may be removed without interfering with the function of the intestine. A **study**

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[1] Zeit. f. klin. Med., Bd. XXXVIII, Hefte 4, 5, u. 6.

[2] Lancet, June 23, 1900.

[3] Lancet, Jan. 27, 1900.

**of absorption** in this case showed a loss of 10.97 % of nitrogen and of 13.91 % of fat. Both are high percentages, particularly that for the fat, and the man showed evidences of imperfect functionation, as after some time it was found that he was losing weight, and he was not able to take solid food. Schlatter believes that the removal of 2 m. of the intestine must be considered to involve danger of imperfect intestinal absorption.

J. K. Johns [1] gives a secondary report of a case in which **30 inches of intestine were removed** by Deaver 5 years ago. The patient had remained well subsequently, and the loss of intestine seemed to have no noticeable influence upon his nutrition.

**Treatment.**—H. S. Upson [2] recommends as a harmless mechanical laxative the use of **white or liquid petroleum** given by the mouth in doses of 2 or 3 ounces. It is said to be well taken and readily borne even by infants.

Overlach [3] claims remarkable results in diarrheas from the use of **fortoin,** a combination of formaldehyd with cotoin. It occurs in the form of tasteless yellow crystals or powder. It is insoluble in water, but readily soluble in alkalies. The dose recommended for adults is about 4 grains 3 times a day. It is claimed that this drug increases the circulation of the bowel-wall, and thus leads to rapid repair of lesions, and that it also acts as an antifermentative and is bactericidal.

Rolley,[4] after investigating the value of **ichthalbin** in intestinal diseases, states that this substance does not produce any harmful results or cause constipation; and that it increases the appetite, lessens the amount of ethereal sulphates in the urine, and improves the catarrhal condition of the bowel or of the stomach in subacute or chronic cases, but is of relatively little value in acute cases. He gives it to children under a year in doses as large as 7 grains; to adults in doses as large as 2 drams in a day.

C. Frohlich [5] reports useful results from the treatment of diarrhea with **tannopin,** which is 81 % tannin and 13 % urotropin. The dose for adults is from 1½ gm. to 4 gm. a day; for children, 0.75 gm. to 2 gm. a day.

T. C. Test [6] reports valuable results from the use of **creasote** for intestinal fermentation. In one case epileptic attacks seemed to be immediately due to gastro-intestinal disturbance, and the use of creasote overcame this.

## ENTEROPTOSIS.

Godart-Danheiux [7] presents some interesting observations of the **relations between movable kidney and enteroptosis.** He has made a series of 871 observations on patients, most of whom showed disturbance of nutrition. In 268 male subjects he found movable kidney 6 times; once it was bilateral and in other instances on the right. In 603 women he found 212 cases of nephroptosis; in 183 cases this was of the right kidney, in 29 bilateral, and only once was the left kidney alone affected. The general percentage of nephroptosis in men was 2.33; in women, 35.1. In the 603 women he found enteroptosis 178 times; that is, in 29.5%. He also notes that he found ptosis of the liver in 3.8% of the women. He presents a study of the cases in their relation to age and to pregnancy, and states that the most common cause of enteroptosis is a series of pregnancies, these acting through the diminution of the abdominal tension. Age accentuates the tendency to enteroptosis even in nulliparæ; on the contrary, neither repeated pregnancy, advanced age, nor similar conditions have any discoverable influence upon the production of movable kidney. He considers enteroptosis to be always produced by a diminution of abdominal tension. It is not always accompanied by nephroptosis. There is no parallelism between the frequency of the two conditions at different ages and under the influence of different factors. He thinks, therefore, that they are due to different causes.

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[1] Med. News, Dec. 2, 1899.

[2] Phila. Med. Jour., July 22, 1899.

[3] Centralbl. f. innere Med., Mar. 10, 1900.

[4] Münch. med. Woch., April 24, 1900.

[5] Münch. med. Woch., July 18, 1899.

[6] N. Y. Med. Jour., April 7, 1900.

[7] Gaz. hebdom. de méd. et de chir., Feb. 18, 1900.

B. Stiller[1] considers that his **sign of enteroptosis**—that is, movability of the tenth rib—is found commonly in hypersecretion of gastric juice (Reichmann's disease) as well as in enteroptosis. He thinks that hypersecretion is usually, like enteroptosis, dependent upon a congenital predisposition. Most cases of nervous dyspepsia he considers dependent upon enteroptosis, and, in opposition to Glénard, insists that enteroptosis is ordinarily the result of congenital tendencies and is not acquired. There are two degrees of movability of the tenth rib—partial and complete. He believes that the presence of this movability in a case of enteroptosis indicates positively that the enteroptosis is congenital. He considers this costal stigma a strong **indication of a neurasthenic tendency,** and its degree to a considerable extent an indication of the severity of the neurasthenia, and, in enteroptosis, of the degree of this abnormality. If the tenth rib is found movable in a child, he considers that the child will practically surely have neurasthenia in the future, probably associated with dyspepsia and enteroptosis. Nervous dyspepsia and enteroptosis are considered practically identical. The symptoms in other cases are dependent not so much upon the position of the viscera as upon the neurasthenia. The most prominent symptom in most cases, as well as the earliest, is atony of the stomach. In the first place, there is commonly hyperacidity, often followed by hypersecretion. There usually exists atony of the intestine, and he considers that constipation in young people is very frequently a result of enteroptosis, and that enteroptosis is almost as common in men as in women. He thinks that tight lacing, pregnancy, and similar conditions are of little importance in the production of the form of enteroptosis usually seen. The condition arises from a neurasthenic tendency.

E. Meinert[2] disputes the value of movability of the tenth rib as a sign of enteroptosis. In examining 100 women as to the presence of this sign he found it in but one. She was a dyspeptic and anemic patient with myoma of the uterus who had marked gastroptosis. In all other cases the rib was more or less fixed. In 20 men with gastroptosis he found the tenth rib always fixed, and in 100 postmortems on the bodies of 50 men and 50 women he found the costa fluctuans decima only 3 times; 2 of these were in men between 45 and 49 years of age, and the third in a woman of 43. None of these three bodies corresponded in general build to the type which Stiller describes as characteristic.

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[1] Berl. klin. Woch., Aug. 21, Aug. 28, and Sept. 4, 1899.

[2] Wien. klin. Woch., June 4, 1900.

P. Reynier [1] considers the presence of a **painful point in the left hypochondrium** over the false ribs at about the level of the waist a sign of value in indicating the presence of enteroptosis. This point is just about the position of the angle between the transverse and the descending colon. The pain and tenderness are the result of a kink at this point, and are chiefly due to the consequent distention with gas.

R. L. Jones and T. A. Clinch [2] describe a case in which there was disease of the heart, and also what proved to be splanchnoptosis. A mass running across the abdomen was felt in the epigastrium. This was of doubtful nature. At necropsy it proved to be the **pancreas, which was laid bare** by the gastroptosis. They consider that this condition has not been discovered before, and is of importance clinically. [The fact that the pancreas is palpable in gastroptosis has been repeatedly noted before, however, and we have had opportunity to observe this in a considerable number of cases.]

W. F. Hamilton,[3] in a description of 6 cases of enteroptosis, makes the interesting statement that in one case the transverse band which is so frequently felt a little above the umbilicus in cases of gastroptosis was present, and was found upon operation to be the pancreas.

## PHANTOM TUMOR.

R. H. Fitz [4] directs attention to the fact that descriptions of phantom tumors have been relatively few in recent years, while reports of so-called **idiopathic dilation of the colon** are increasing in number; the latter cases exhibit a series of characteristics which closely resemble those of phantom tumor. He describes instances of this, the last of which was one in which all the characteristics of phantom tumor, even to reduction under anesthesia, were seen in an elderly woman of neurasthenic type. In the presence of appearances of phantom tumor dilation of the colon should be sought for. Fitz agrees with Treves in recommending operation in these cases.

## CARCINOMA.

I. Boas,[5] in discussing his experience with **carcinoma of the large intestine,** states that he has seen 15 certain cases, of which 12 occurred in men and 3 in women. In 6 instances tumor occurred at the cecum, twice at the hepatic and splenic flexures, once in the descending colon, and 4 times in the sigmoid flexure. In 8 cases a tumor was distinctly palpable. As to the subjective symptoms, he divides them into 4 groups: In the first there are no local symptoms, and if a diagnosis is made, it must be established on the basis of the cachexia. In the second group there are obscure local symptoms. In the third group there are pronounced local symptoms of stenosis, with recurring attacks of colic and diarrhea; when the tumor is situated low down, there is also usually tenesmus of both rectum and bladder. In the fourth group he puts those cases in which, in the midst of health, there is the sudden appearance of more or less complete obstruction of the intestine. Boas has in most cases not noticed the marked movability of the tumor in carcinoma of the colon which has been insisted upon by some other writers. He has often observed, however, that the tumor is very easily palpable at certain times, and at other times almost impalpable, so that it is always necessary to palpate after having the bowels cleared out, and after filling the bowels with air and water. The most difficult diagnosis is from tuberculosis, inflammatory infiltrations, and benign tumors. In differentiating from tuberculosis it is important to remember that in most cases of tuberculosis of the bowel there is a marked diazo reaction, while in cancer of the bowel this is practically never present. The most important diagnostic signs of carcinoma of the bowel are those of stenosis, and particularly important is **visible and palpable peristalsis.** In 2 cases Boas determined that the active peristalsis was of the small intestine by feeling coils of contracted bowel, which were evidently small intestine, by examination *per vaginam* during the attack. The sudden appearance of peristalsis in the coil of intestine, with lightning-like disappearance and reappearance, is a sign that Boas has repeatedly noticed, and considers absolutely diagnostic of stenosis. He investigated the condition of the stomach-contents in his cases, and found it very variable. Free HCl may be present or absent. In 2 cases he saw hemorrhages from the stomach. He insists that the condition of the bowel movements is extremely variable, and that in many cases the stools are normally formed. The presence of blood and pus in the bowel movements is very important. In one instance Boas saw a complicating membranous enteritis—a complication which had not previously been described. Of the 15 cases, 12 were operated upon. In 5 cases resection was done, and 3 died; while of the remaining 2, one is well 5 years after the operation, and the other still well after 5 months have passed. More palliative operations are less satisfactory, but give much comfort, and Boas recommends operation very decidedly. Resection is most satisfactory, if feasible, and is often followed by a long period of freedom from recurrence or metastasis.

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[1] Jour. de méd. de Par., April 1, 1900.

[2] Edinb. Med. Jour., Nov., 1899.

[3] Montreal Med. Jour., Sept., 1899.

[4] Am. Jour. Med. Sci., Aug., 1899.

[5] Deut. med. Woch., Feb. 15 and 22, 1900.

A. Descot and L. Beriel [1] report an interesting case of **perivaterian cancer of the duodenum** which involved the biliary passages and the head of the pancreas without causing progressive icterus. The case was a woman who had suffered from colicky attacks and diarrhea. The signs when she came under observation pointed to carcinoma of the stomach, but the autopsy showed cancer of the duodenum, which involved probably the whole wall of the bowel at the ampulla of Vater as well as above and below, and included the head of the pancreas. The walls of the bile-duct were thickened and somewhat rigid, and microscopically this proved to be carcinomatous tissue, but the bile passages were dilated instead of contracted. The authors attribute the dilation to the loss of elasticity in the walls, and consequent passive distention from accumulation of bile. From the absence of obstruction of the bile-duct, and apparently of the pancreatic duct also, they decided that the carcinomatous growth began about the ampulla, and not at its orifice.

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[1] Rev. de méd., Aug. 10, 1899.

## DISEASES OF THE PERITONEUM.

A. James [1] describes a case of effusion into the peritoneum and pleura in which there was an evident **communication between the peritoneum and pleura,** since tapping from the peritoneum or pleura reduced the fluid in both cavities, and the abdominal cavity seemed to be more thoroughly emptied by tapping the pleura than by direct tapping.

T. T. Fisher [2] reports the case of a woman of 38 who after being chilled had swelling and pain in the abdomen with general tenderness and evidences of effusion. There was also blood and mucus in the stools. The patient died suddenly, and autopsy disclosed a large **hemorrhagic abdominal effusion,** with extreme congestion of the intestine in the distribution of the superior mesenteric vein, but no thrombosis of the vein. The veins of the other abdominal organs were very much distended, but there was no evidence of antemortem clots. The spleen was large and there was a much enlarged lymphatic gland alongside the trachea. Cultures from this and from the spleen showed the colon bacillus, and the case was probably one of infection, perhaps from the alimentary canal.

Zuppinger [3] draws attention to the fact that it may at times be possible to **mistake purulent meningitis for a perforation peritonitis,** and reports a case in point which occurred in a child 3 years and 9 months of age. There was stricture of the esophagus following ingestion of caustic potash, and a bougie was introduced in treatment. The child for some hours had no sign of any resultant injury, but was suddenly taken with headache, severe pain in the abdomen, vomiting, and chills, and the abdomen was very sensitive. The child died some hours later—it was thought of perforation peritonitis; but autopsy showed that the cause of death was purulent meningitis.

F. Widal and P. Merklen [4] discuss the **diagnosis** between **chylous ascites** and other conditions in which there is ascites which resembles the chylous form, but in which the milky appearance is due to the presence of lymph. They state that the diagnosis may be made by investigating the character of the leukocytes in the fluid. The mere presence of fat in considerable quantities does not demonstrate that the substance is chyle, as large amounts of fat may be present under other circumstances. In the case which they report, in which 9 liters of milky fluid were removed from the abdominal cavity just before death, they found that there were large numbers of leukocytes in the fluid (about 100,000 per cc.), and that among these leukocytes there were no polymorphonuclear, eosinophile, or basophile cells, the only elements found being lymphocytes and large mononuclear cells. Red cells were absent. Hence it was concluded that the ascites was of lymphatic origin. The fluid contained 1.450 gm. of fat per liter, but this large amount of fat led to confusion in the diagnosis.

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[1] Brit. Med. Jour., July 29, 1899.

[2] Lancet, Jan. 6, 1900.

[3] Wien. klin. Woch., Aug. 24, 1899.

[4] Gaz. des Hôp., Feb. 23, 1899.

F. Micheli and G. Mattirolo [1] describe 4 cases of ascites in which there was an appearance of the presence of chyle, but fat was absent, and hence the condition was not chylous ascites. They believe that the **turbidity is probably due to** the presence of **lecithin** in suspension.

J. H. Croom [2] describes the case of a woman of 39 who, together with increasing weakness and emaciation, had an exudate in the abdominal cavity which, when aspirated, was of a pus-like appearance and contained 2 % of fat, with many degenerating leukocytes. Death ensued, and the autopsy showed cancerous infiltration of the stomach, intestines, mesentery, and main bile-ducts. The lacteals were occluded and the **receptaculum chyli was pressed upon** by a cancerous mass.

A. H. Smith,[3] in discussing the possibility of the diagnosis of **obliteration of the receptaculum chyli** or of the thoracic duct, refers to the case of President Garfield, and to the fact that the emaciation in the last few weeks of his life was astonishing. This Smith believes was due to the fact that, as disclosed at autopsy, the thoracic duct was entangled in a mass of exudate which had compressed and probably concealed the duct. Another case is referred to in which a mass situated in front of the first and second lumbar vertebræ caused obliteration of the receptaculum chyli. In this case there was also extremely rapid emaciation. Smith believes that if there are symptoms of disease in the neighborhood of the stomach, with very striking emaciation, and especially with a tendency to copious, pale, and fluid bowel movements, obliteration of the duct or receptaculum should be considered.

## DISEASES OF THE LIVER.

**Icterus.**—H. Neumann [4] has made an extensive study of so-called **simple catarrhal icterus,** using as a basis the 215 cases that had been seen in Senator's clinic between 1892 and 1896. Over 40 % occurred within the first decad of life, while nearly 27 % affected those in the third decad. After the first five years of life males were much more frequently affected than females. The cases were much more numerous during the first and fourth quarters of the year; that is, in the fall and winter months. These facts are similar to those observed in relation to epidemic infectious jaundice, and are used to emphasize Neumann's opinion that there is no demonstrable difference between so-called simple catarrhal jaundice and epidemic infectious jaundice. Further proof of this view is found in the fact that although the jaundice showed no relation to the water-supply, it occurred in certain definite portions of Berlin, and tended to appear repeatedly in the same region when once it had shown itself in that portion of the city. Also, there are likely to be frequent relapses in jaundice, which are much more readily explained by attributing the whole to infection and reinfection than by considering it as due to a simple catarrhal condition.

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[1] Wien. klin. Woch., Jan. 18, 1900.

[2] Lancet, June 23, 1900.

[3] Med. Rec., Dec. 2, 1899.

[4] Deut. med. Woch., Aug. 31, 1899.

W. Wenthall [1] describes a case of **icterus gravis** which occurred in a man of 29. His illness began with weakness and vomiting, and on the fourth day he had a convulsion. Jaundice appeared 2 days later, and 2 days after this he had another convulsion. The jaundice deepened and became very dense, but the liver did not enlarge and the spleen was not palpable. Petechial hemorrhages appeared, nervous symptoms became pronounced, the temperature was often high, and he died in coma on the thirteenth day. The urine had contained albumin and casts, and the blood showed a leukocytosis of 21,000. The differential count showed 18% of myelocytes, with a diminution in the mononuclear elements. The autopsy showed the liver to be bile-stained, but otherwise very little changed; the spleen was large and soft, and bacteriologic examination showed a number of micro-organisms, Staphylococcus pyogenes and Bacillus pyocyaneus being isolated. Bacillus pyocyaneus was also found in the liver in large numbers, and the process was thought to be one of pyocyaneus bacillemia. The man was a worker in a skin-curing yard, and might readily have been infected in his occupation.

**Cirrhosis.**—W. B. Cheadle,[2] in discussing cirrhosis of the liver, states that **contracted liver** is practically **always alcoholic in origin.** If a primary stage of enlargement occurs, it must be attributed to engorgement. This he has observed, but he does not consider hypertrophic cirrhosis a primary stage of the atrophic variety. **Ascites** occurred in 17 of 23 fatal cases of trophic cirrhosis, and usually indicated that death would occur within a few months. Jaundice is relatively frequent in atrophic cirrhosis, contrary to the common teaching. He saw marked jaundice in 8 of 53 fatal cases of cirrhosis of all kinds, and in 4 of these the liver was much contracted. He has for 16 years watched cases of cirrhosis carefully without being able to discover any one that corresponded typically with Hanot's description. **Jaundice** is by no means constantly present in hypertrophic cirrhosis. It was a marked symptom in only 4 of 17 consecutive fatal cases. Also ascites is by no means uncommon in hypertrophic cirrhosis. Enlargement of the spleen is an especial feature of this form. Ascites was present in 16 of 25 fatal cases. The **spleen was enlarged** in the majority of all forms of alcoholic cirrhosis. Pain, fever, and nervous symptoms are often said to be characteristic of biliary cirrhosis, but may be seen in any form, particularly in syphilitic cirrhosis. Hypertrophic cirrhosis is usually chronic, but there are acute cases reported. **Syphilitic cirrhosis** is likely to be mistaken for atrophic alcoholic cirrhosis, and signs of perihepatitis are some evidence in favor of syphilitic origin. It is notable in many cases that the appearance of syphilis, even postmortem, may be very slight. There is usually decided perihepatitis; no local peritonitis. In many instances one obtains no history of syphilis. He notes the affections of other organs connected with the cirrhosis. In 36 fatal cases **the heart** showed fatty degeneration in 6, myocarditis in 1, probable fatty changes in 8 others. **The kidneys** were cirrhosed in 26 of 53 cases. **The spleen** was enlarged in 23 cases, 11 of which were atrophic cirrhosis, 12 hypertrophic. **The pancreas** was examined in 14 cases. It was cirrhosed in 3, abnormally hard in 10, and in the remaining case was large and soft. **Erysipelas** has frequently been noticed with cirrhosis, and Cheadle has seen 5 fatalities in this disease from erysipelas. He believes that it is impossible to make any absolute classification of cases of cirrhosis. As to **prognosis,** he states that ascites is a very unfavorable sign; and when it appears, the case is not likely to last more than a few months. The prognosis is better in the hypertrophic form than in the atrophic, and the general appearance of favorable cases is that the liver is large, the subjects are usually young and well nourished, and, as a rule, they have been hard drinkers or are syphilitics. He concludes his lectures by considering the **treatment.** It is essential to check the process of the disease by excluding alcohol and all stimulants, and also forms of food which embarrass the liver, such as fats and sugar. Cheadle insists that cirrhosis is too commonly considered a mere obstructive disease, while as a matter of fact one of the serious elements is the disturbance of the functions of the liver in aiding absorption, assimilation, and elimination. **Predigested foods** should be used if there are evidences of disturbed nutrition. Mild laxatives may be used, and mercury and iodids are always in place in syphilitic cases, and even in others may frequently do good. Digitalis is often necessitated by weakness of the heart. Ascites is the symptom which demands the most active intervention. Purgatives are likely to be dangerous. Diuretics are commonly useless. Restriction of fluids may do some good, but Cheadle believes that **paracentesis** should be undertaken early and repeatedly if necessary. He reports a series of cases in which paracentesis was undertaken early in the disease, and years of comparatively good health followed. In one case the woman was still living after 12 years, and seemed in good health except for the existence of a large, hard liver. A number of cases almost as favorable as these are reported also. Heart weakness often determines the occurrence of ascites, and in such cases the prognosis is usually better, because cardiac debility can be improved. Cheadle also suggests that perhaps the reason for the occurrence in some cases of a hypertrophic cirrhosis and in others of an atrophic form, is that in the first class the heart is weak and there is stagnation of blood in the small vessels, and the cirrhosis tends to occur in this region as it does in hypertrophic cirrhosis; also in such cases the alcohol will have its most active effect where the stagnation is most marked—in other words, in the fine vessels.

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[1] Lancet, Dec. 9, 1899.

[2] Lancet, Mar. 31, and Brit. Med. Jour., April 7 and 14, 1900.

A. Gilbert and L. Fournier [1] describe a case of **biliary cirrhosis** with extreme enlargement of the spleen. They believe that there is a class of cases characterized by chronic icterus with great splenic enlargement, and with only moderate enlargement of the liver. This differs from so-called primary splenomegaly in the prominence of the hepatic symptoms, particularly icterus. They consider it to be probably due to ascending infection of the biliary passages.

Milian [2] reported a case of **hypertrophic biliary cirrhosis** which began with progressive enlargement of the spleen and disturbance of digestion. There were in the early stages no other signs than the great enlargement of the spleen, which led to the suspicion of splenic leukemia, but the blood was negative. Subsequently icterus appeared, which became more pronounced, the liver enlarged, and the bile appeared in the urine. The man died of repeated hematemesis. The autopsy showed that it was hypertrophic cirrhosis of the liver without ascites, death being due to rupture of the esophageal varix. The spleen weighed 1900 gm. and the liver 2070 gm. There was a striking enlargement of the abdominal glands. The blood did not coagulate.

W. Osler [3] reports 2 cases of **hypertrophic cirrhosis** of the liver **with hemochromatosis.** Neither patient presented glycosuria. The first was a man of 48 who had pigmentation chiefly about the nipples and genitalia, with slighter general pigmentation and marked bronzing of the hands, wrists, and legs; the mucous membranes were free. He had repeated attacks of purpura and urticaria. The spleen and liver were much enlarged. The urine gave decided reaction for indican and iron, but no bile was present. An excised portion of the skin showed ochre-colored pigment granules in the cells of the sweat-glands. The second patient had had malaria and a tropical fever. He had marked enlargement of the liver and spleen, with increasing pigmentation of the face and hands. It was suspected that there was a tumor of the liver, but operation showed only enlargement and deep pigmentation.

J. Dougall [4] describes a case in which after remittent fever the man showed signs of syphilis. After this he had enlargement of the liver with ascites, and great enlargement of the spleen. The urine contained a little albumin and bile, but was otherwise negative. The severity of the general symptoms and the rapidity with which the ascites returned after tapping led him to believe that the case would be inevitably fatal; but after 26 tappings had been done, and, in all, 46 gallons of fluid had been removed, the patient began to improve, and ultimately left the hospital well except for some albuminuria. The case was thought to be one of **simple induration** of the liver.

R. B. Preble,[5] after studying the reports of 60 cases of **fatal gastro-**

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[1] Gaz. des Hôp., May 25, 1900.
[2] Gaz. des Hôp., April 6, 1900.
[3] Brit. Med. Jour., Dec. 9, 1899.
[4] Edinb. Med. Jour., Oct., 1899.
[5] Am. Jour. Med. Sci., Mar., 1900.

**intestinal hemorrhage** caused by cirrhosis of the liver, decides that it is a somewhat infrequent, though not rare, complication of cirrhosis. It commonly complicates the atrophic variety, but may occur in the hypertrophic form. The first hemorrhage is fatal in one-third of the cases. Hemorrhages have been known to continue for as long a period as 11 years. Diagnosis was made in only about one-third the cases at the time of the first hemorrhage. In 80 % of these cases esophageal varices were present, and in over half of this 80 % there was evident rupture of these varices, and probably rupture could be found in many other cases with proper investigation. Fatal hemorrhages occur, however, when varices are not present, and probably may be due to rupture of numerous capillaries in the gastro-intestinal mucous membranes. Commonly other symptoms of cirrhosis are present before the hemorrhages occur, but hemorrhages may be the first symptom. In only 6 % of the cases in which esophageal varices were present was the cirrhosis of the form described as typically atrophic; that is, with enlargement of the spleen and of the subcutaneous abdominal veins.

P. Spillmann and J. Demange[1] gave **hepatic extract** in 10 cases with various affections of the liver. They observed an increase in the amount of urine secreted and a corresponding increase in the amount of urea and phosphoric acid, with a decrease in albumin and urobilin. The results were unexpectedly favorable in atrophic cirrhosis, and some improvement was observed in passive congestion of the liver and even in cancer, but no results were noticed in hypertrophic cirrhosis.

**Floating Liver.**—M. Einhorn,[2] in discussing floating liver and its clinical significance, records that 30 such cases have come under his observation out of a total of 804 patients seen within 5 months. It was seen in about 3.7 % of cases showing disturbance of digestion. It appears much more frequently in the female than in the male. He divides the condition symptomatically into 5 classes: the first class is composed of cases which show no symptoms, the second of cases with indefinite indigestion, the third of instances of hepatalgia, the fourth of cases of hepatic colic, and the fifth of asthmatic cases. He illustrates these forms by clinical histories. The chief causes of the condition which he mentions are tight lacing, frequent pregnancies, heavy lifting, carrying heavy loads, falls, and injuries.

**Abscess.**—Kelsch[3] describes a series of cases of abscess of the liver, directing especial attention to the fact that the diagnosis was confirmed in two of the cases by the use of the **x-rays,** this method of examination demonstrating the fact that the diaphragm was immobile on the right side and that there was a shadow rising to an abnormal height above the liver, and having a somewhat rounded outline. He considers the x-rays a valuable method of determining the existence of subdiaphragmatic collections of pus or of associated disease of the lung.

W. Bain[4] reports his investigations of the **metabolism in** a case of **liver abscess** following dysentery. The man died, and the liver was found to contain 6 large abscesses in the right lobe, so that the organ was largely destroyed. The urea, uric acid, phosphoric acid, ammonia, and alloxur bases were investigated. The general results showed nothing varying greatly from the normal, which was of interest in showing that the functions of the liver were apparently well carried out, even though the organ was so largely destroyed.

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[1] V Congres français de med. interne, 1899.

[2] Med. Rec., Sept. 16, 1899.

[3] Bull. Acad. de méd., Mar. 6, 1900.

[4] Edinb. Med. Jour., Oct., 1899.

J. F. Richardson [1] reports the case of a man of 24 who was taken ill with chill, severe pain in the region of the liver, and vomiting. He soon developed jaundice and enlargement of the liver, and had irregular fever. After about 3 weeks' illness he suddenly expectorated a large quantity of creamy pus, the purulent expectoration continuing for a considerable time afterward, and being accompanied by a progressive decrease in the size of the liver and by subsidence of the constitutional symptoms. Eighteen quarts of pus were said to have been evacuated in 21 days. The patient ultimately recovered entirely except for some lack of physical endurance and a little uneasiness about the liver. The case was apparently one of hepatic abscess.

**Echinococcus.**—Posselt [2] discusses echinococcus of the liver, chiefly in relation to symptomatology and diagnosis. The most important symptoms are enlargement and alteration in the shape of the liver. Enlargement is usually of the right lobe alone. The liver is felt to be hard, and there may be pain and tenderness if the growth is near the surface, but this is not always the case. There is usually marked jaundice, and later frequently ascites, and rather commonly free sweating; there is usually constipation, and at times in the later stages of the disease blood appears in the stools. A striking characteristic is that the body-weight usually does not decrease to any extent; this is very important in diagnosis, for the diseases showing the greatest similarity are carcinoma and hypertrophic cirrhosis, but in these the body-weight is reduced. Cachexia does not come on in echinococcus disease until very late. The appetite is usually good, and may be excessive; there is commonly no fever; the spleen does not enlarge. He recommends puncture if the condition of the tumor favors it, and if there is no evidence of marked inflammatory reaction; but puncture is chiefly useful for diagnostic purposes. A striking symptom which is frequently seen is polyuria. Posselt thinks that this is due to functional hypertrophy of the kidneys. As to the location of the disease, it is very common in Bavaria, Austria, Switzerland, and Würtemberg, but is rare elsewhere. In these parts of the world especially it is dangerous to allow children to come freely into contact with dogs, as the disease is chiefly conveyed from them.

**Malignant Growths.**—T. L. Brunton [3] describes a malignant growth which produced jaundice, somewhat acholous stools, and ascites with enlargement of the liver. After death there was found a growth pressing upon the portal vein and hepatic duct. There was a ruptured ovarian cyst, and the abdominal cavity was covered with new growths. The growth in the liver was probably secondary to the ovarian growth.

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[1] N. Y. Med. Jour., April 21, 1900.

[2] Deut. Arch. f. klin. Med., Aug. 18, 1899.

[3] Edinb. Med. Jour., Oct., 1899.

## DISEASES OF THE BILIARY PASSAGES.

J. H. Keay,[1] from his own experience, and from careful questioning of a large number of cases, has concluded that the common teaching as to the **seat of pain in biliary colic** is erroneous. He finds that the pain usually begins in the back near the tenth or eleventh dorsal vertebra, that it may last for a considerable period preceding an outbreak of colic, and that it is often mistaken for lumbago and sometimes for spinal trouble. It then works round to the front as the attack comes on, and then agonizing pain is felt in the right or left hypochondriac region, and sometimes lower in the abdomen. Pain may be felt above the right nipple, but he finds that it practically never is noticed in the shoulder. He has sometimes felt a gliding sensation in the back to the right of the two lower dorsal vertebræ as the stone passed into the duodenum. The pain that *begins* in the right hypochondrium is, in his experience, not associated with the passage of gall-stones, but is due to distention of the gall-bladder or to localized peritonitis. The administration of a few whiffs of chloroform often controls the attack without producing unconsciousness.

S. Droba,[2] as a demonstration of the relation between **typhoid fever and cholelithiasis,** describes a case which was operated upon for cholelithiasis, and in which the gall-bladder was entirely removed because of extensive adhesions and great thickening. A portion of the liver was necessarily resected with the growth of the bladder. Bacteriologic examination of the gall-bladder showed the presence of typhoid bacilli. There were numerous gall-stones in the gall-bladder, and cultures from the interior of these showed typhoid bacilli also. An attack of typhoid fever had occurred 17 years previously, and the biliary colic had come on after this.

J. M. Da Costa[3] believes that more careful study will show that **recovery from cholecystitis in typhoid** fever is more frequent than is commonly thought, though the complication is likely to be grave. He reports 3 recoveries from this affection in cases which were mild, but which seemed to be undoubtedly cholecystitis. One case had severe jaundice, while in the others it was only slight. The treatment he recommends is mild doses of calomel and the local use of ice, poultices, and counterirritation, giving morphin if necessary, and supporting the circulation with strychnin and digitalis.

P. Clairmont[4] describes a case of **cholangitis** and abscess about the gall-bladder in a woman of 79 in which the infection was due to **Friedländer's pneumobacillus.** He observed that these organisms stained with Gram's method in tissues when they have been fixed in Müller-formol, while they do not stain by this method in cultures.

F. A. Packard[5] reports a case of **acute cholecystitis** in a man of 58 which occurred after an attack of **influenza,** and in which it was thought possible that the influenza had etiologic relation to the cholecystitis.

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[1] Brit. Med. Jour., April 14, 1900.

[2] Wien. klin. Woch., Nov. 9, 1899.

[3] Am. Jour. Med. Sci., Aug., 1899.

[4] Wien. klin. Woch., Oct. 26, 1899.

[5] Phila. Med. Jour., Feb., 1900.

T. J. Maclagan and F. Treves [1] report 3 cases in which there were symptoms of gall-stones and in which operation was undertaken and the symptoms were found to be due simply to a **movable right kidney** causing pressure on the bile-duct. Fixation of the kidneys caused the disappearance of all symptoms.

Pouchet [2] advises the use of **amyl valerianate** in cholelithiasis because it has a solvent action on cholesterin. He gives it either in capsules or in emulsion.

## DISEASES OF THE PANCREAS.

J. M. Anders [3] reports cases of **pancreatic hemorrhage.** The first, a man 28 years old, was taken with severe epigastric pain with diarrhea, and afterward severe constipation. Ten days later there was jaundice. He had severe pain about 2 hours after taking food, and ulcer of the duodenum was suspected. Death occurred with symptoms of collapse from internal hemorrhage, and postmortem showed the smaller peritoneal cavity filled with blood. There was carcinoma of the pancreas and of the surrounding lymphatic glands, the hemorrhage being thought to have resulted from the changes caused by the carcinoma. In the second case there was a history of injury 2 years previously, and subsequent frequent pain upon exertion or after eating, and the final illness came on with severe epigastric pain, collapse, and extreme tenderness in the lower left side of the epigastrium. Blood was found about the pancreas at the postmortem, and the organ was hemorrhagic. Anders presents a table of 40 cases of pancreatic hemorrhage.

Dupuy [4] describes a case in which sudden death occurred from hemorrhage into the pancreas. The autopsy disclosed marked arteriosclerosis; the splenic artery showed pronounced atheroma, and it was decided that the hemorrhage into the pancreas resulted from the **atheroma of the splenic artery and its branches.** The man had a right-sided pleural effusion—a condition which is common in arteriosclerosis.

J. C. Uhthoff and E. F. Maynard [5] report a case of pancreatitis which occurred in a man of 77. The symptoms were the usual ones, and death occurred within 3 days, the only peculiarity of the attack being a daily onset of severe collapse with partial recovery of strength. At the autopsy the head of the pancreas was found to be the size of an orange, there was **one large hemorrhage and several smaller ones,** and the capsule was much thickened.

M. B. Schmidt [6] describes a case of fat-necrosis which occurred in a man who had been crushed between the bumpers of railway cars, and who died 2½ days afterward with the signs of intestinal obstruction. There was very little hemorrhage in the **pancreas,** but the organ was

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[1] Lancet, Jan. 6, 1900.

[2] Jour. de Praticiens, No. 45, 1899.

[3] Jour. Am. Med. Assoc., Dec. 2, 1899.

[4] Jour. de Praticiens, Sept. 16, 1899.

[5] Brit. Med. Jour., June 3, 1900.

[6] Münch. med. Woch., May 8, 1900.

**severely torn.** The fat-necrosis was believed to be due to the escape of pancreatic secretion.

J. A. Scott [1] reports a case of **acute gangrenous pancreatitis** with necrosis, which occurred in a woman of 64 who for years had suffered from attacks of gastro-intestinal trouble, chiefly evidenced by pain and distention of the stomach. The attack which caused her death began with violent abdominal pain, tenderness, and vomiting; this was accompanied by diarrhea, and she soon became delirious and practically maniacal. Nothing could be felt in the epigastrium. The Widal test was positive. The diagnosis was very doubtful. The patient died on the fifteenth day of her illness, and the postmortem showed marked fat-necrosis in the omentum, a localized peritonitis in the epigastric region, and enlargement, softening, and abscesses of the pancreas; there were hemorrhagic-looking areas throughout it, and in some places it was entirely gangrenous. Scott has investigated the postmortem records of the Pennsylvania Hospital since 1876, and found but one other record of acute pancreatitis.

H. W. Jacob [2] reports an attack in a boy of 10 which **complicated mumps,** and which was considered to be **acute pancreatitis.** As the swelling of the parotids disappeared the patient complained of severe abdominal tenderness with some pain, and a mass was felt in the region of the pancreas. The tenderness and mass gradually disappeared under treatment, and the boy recovered.

J. Israel [3] reports an extremely interesting case in which a very **movable tumor proved to be the pancreas.** When the patient was lying upon her back, the tumor presented in the epigastrium; upon change of posture, it would move from the middle line to the anterior axillary line, and vertically from the level of the eighth rib to the level of the twelfth. The patient was an unmarried woman of 39 who had been well until she had an attack with the symptoms of gastritis. This was followed by a feeling of pressure in the stomach, with rapid emaciation, and her health became very much impaired. Operation showed that there was a pancreatic cyst with a long pedicle, and that the whole pancreas was movable. Israel also describes a case of extremely movable pancreatic cyst which was interesting because of the mobility of the cyst and because the fluid contained in the tumor showed the absence of ferments; ferments appeared, however, a few days after operation in the fluid drawn from the fistula. He also describes a case in which a single aspiration of a pancreatic cyst resulted in complete cure. The case of movable pancreas is the first reported.

H. Keitler, [4] reports a cystic **tumor of the pancreas** which was pedunculated, and **notable for the freedom of its movement** in the abdomen. It could be easily displaced upward under the ribs, to the left, or well downward, and it moved readily with respiration. It resembled clinically a cyst of the kidney. E. Ždarek, [5] in examining the fluid from this cyst, found that it was somewhat unusual. It had a neutral reaction, the specific gravity was 1007, and there was less than 1% of albumin. Urea, cholesterin, fatty acids and fats, and a diastatic ferment were found, but no proteolytic or fat-splitting ferment could be demonstrated.

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[1] Am. Jour. Med. Sci., Oct., 1899.

[2] Brit. Med. Jour., June 23, 1900.

[3] Deut. med. Woch., May 31, 1900.

[4] Wien. klin. Woch., July 20, 1899.

[5] Ibid.

Günther [1] describes a case of **carcinoma** of the pancreas in which there was **no glycosuria** and **no apparent excess of fat** in the feces. A lack of these symptoms is usually noticed in pancreatic disease, and is considered to be due to the situation of the growth. [There is not any strong evidence that pancreatic disease causes much decrease in the absorption of fats. There is better evidence that it causes more or less complete loss of the fat-splitting action.]

## FOOD-POISONING AND GASTRO-INTESTINAL INTOXICATION.

Nason [2] discusses an **outbreak of food-poisoning** which he has recently observed at Nuneaton. The histories of 42 cases were investigated. The affection was characterized by pain in the abdomen, looseness of the bowels, vomiting, collapse, and pains or cramps in the legs, together with hot and cold feelings. It was found that those affected had eaten a preparation of pig entrails called chitterlings, and it was found also that all the cases occurred in persons who had eaten the chitterlings without having re-boiled the food after the time of its first preparation, while others who had eaten the same dish after having boiled it subsequent to its first preparation showed no symptoms. It was therefore thought that the outbreak was due to infection of the chitterlings after the preliminary preparation. There was an extremely long interval between the time that the food was taken and the onset of the symptoms, and during this time the subjects of the affection were in good health. It was therefore considered probable that the symptoms were produced by the development of bacteria in the body after the food was ingested, the bacteria—probably Bacilli enteritidis—having been introduced with the chitterlings. A bacteriologic examination could not be carried out.

M. L. Hughes and C. W. R. Healey [3] report an **acute epidemic** of **gastro-enteritis,** which they attributed to food-poisoning, though the definite source of the poisoning could not be determined. Many of the persons affected had eaten portions of a certain cheese which contained considerable amounts of tyrotoxicon, but some of the subjects had not partaken of the cheese. The symptoms were pains in the limbs, headache, gastric pain, anorexia, nausea, and vomiting, with constipation, the latter being followed by diarrhea with slate-colored stools, the stools containing altered blood. The temperature was often high, there was profuse sweating and great thirst, and the urine usually contained albumin and casts. Blood was vomited in 2 cases. In severe cases there was jaundice, which appeared after the first few days, and there were marked nervous symptoms. The cause of death in the fatal cases was heart failure. Three fatal cases showed severe congestion, with inflammation and edema of the stomach and small intestine, and enlargement of the solitary glands. Peyer's patches were unaffected. There was no ulceration and the colon was not involved. The liver was enlarged and showed fatty degeneration. The kidneys were enlarged and congested.

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[1] Deut. Arch. f. klin. Med., Feb. 6, 1900.

[2] Brit. Med. Jour., Sept. 23, 1899.

[3] Lancet, Nov. 4, 1899.

T. Zammit[1] describes a series of cases of **milk-poisoning** in Malta. Seventeen persons, who lived in 5 different houses, were attacked with the symptoms of severe gastro-enteritis associated with collapse about 3 hours after drinking milk. Those who had boiled their milk did not become ill, and the illness occurred only in those who had been served with milk from a can, and was not seen in those families whose milk was served directly from goats, though supplied from the same milkman. The goats were found to be healthy, but the **cans were examined,** and were found to contain a bacillus corresponding to **Bacillus enteritidis sporogenes.** This demonstrates the importance of investigating the condition of the cans and of keeping them thoroughly cleansed and disinfected.

C. Harrington[2] discusses the **diagnosis between food-poisoning and metallic poisoning,** particularly the diagnosis between food-poisoning and arsenic-poisoning. Both begin in very much the same way, with severe gastro-intestinal irritation; but the symptoms in the case of ptomain-poisoning are often delayed from a few hours up to a number of days. In the case of food-poisoning a number of people are likely to be affected at the same time. The differences in the symptoms are chiefly that in arsenic-poisoning there is likely to be dysphagia because of pain, in ptomain-poisoning because of paralysis of the constrictors of the pharynx; and in ptomain-poisoning there is often extreme prostration, practically amounting to paralysis, and marked dilation of the pupils.

W. P. Cones[3] describes a case of what he terms **ptomain-poisoning** which occurred in a man of 26 shortly after a mixed meal. The man fell suddenly, and was observed to be profoundly collapsed, but physical examination was negative. He had, however, considerable pain over the heart, but improved somewhat, and after vomiting and evacuation of the bowels, seemed to be entirely well.

A. Robin[4] reviews the whole question of the existence of **gastro-intestinal intoxication,** concluding that there is absolutely no evidence that gastric toxins exist, that they have never been isolated, and that it is possible neither to deny their existence nor to show that they exist. The whole theory of autointoxication of digestive origin rests purely upon analogy, and there are no definite details known upon which one could base therapeusis. He believes that the attempts at antisepsis of the stomach by administering drugs have been almost valueless so far as their influence upon so-called autointoxication is concerned. He does not deny that there is such a condition, but claims merely that there is no proof of its existence.

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[1] Brit. Med. Jour., May 12, 1900.

[2] Boston Med. Jour., Dec. 14, 1899.

[3] Boston M. and S. Jour., Jan. 25, 1900.

[4] Gaz. hebdom. de méd. et de chir., Mar. 29, 1900.

C. A. Ewald [1] gives a general discussion of **autointoxication**, dividing it into 2 general forms—that produced by intoxication from the intestinal tract, and intoxication from the substances produced within the tissues themselves. Examples of the first form are the nervous phenomena in digestive disturbances, dyspeptic asthma, some of the anemias, etc.; and of the interstitial variety, uremia, diabetic coma, myxedema, leukemia, etc. Ewald considers that the study of the **toxicity of the urine** is subject to great error, because the urine is not isotonic with the blood, the quantities necessarily used are large, the injection is done rapidly, and normal urine contains a greater or less amount of toxic substances. The only conditions which he thinks may be properly called autointoxications are diabetic coma and the rare cases of intoxication by hydrogen sulphid. He considers the whole subject of autointoxication extremely obscure.

F. Forcheimer and R. W. Stewart [2] report their results from a series of experiments concerning **methods of determining the toxicity** of human urine and the value of the various methods. They decide that fresh urine, when injected immediately, gives good results, as does fresh urine which is immediately filtered. Boiling the urine and then injecting it is not a useful method. It is not known in what way boiling reduces its toxicity or alters the effects of ferments or other substances. The addition of boric acid to the urine does not give trustworthy results. Boiling urine immediately, and then injecting it after a considerable period, caused a large fatality, probably because the spores of bacteria were not killed with the bacteria. One can not use urine which has been kept for longer than 24 hours with trustworthy results; injections of old urine caused an extremely high mortality. The results obtained by determining the toxicity of urine have an error of from 25 % to 50 % in individual cases. The toxicity of the urine seems to be due to substances which result from the action of bacteria, and while other toxic substances may be present, it has not been proved that the toxicity is not entirely due to bacterial action.

C. Posner and M. Bertran,[3] in discussing the **fallacies in the investigation of the toxicity** of the urine, note that the quantity used is often excessive. A much more important point than this is the fact that the isotonicity of the urine is very different from that of the blood, the urine containing a much greater proportion of salts than the blood; this is likely to result in great damage from the marked osmotic interchanges which take place. They consider the greater part of the toxicity of the urine due to the difference between its molecular concentration and that of the blood. If urine is made isotonic with the blood, it is not more toxic than an isotonic sodium chlorid solution. This suggests that uremia may be due to alterations in molecular concentration, but the results of the investigations of this question have been somewhat contradictory.

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[1] Berl. klin. Woch., Feb. 12 and 19, 1900.

[2] Am. Jour. Med. Sci., Sept., 1899.

[3] Berl. klin. Woch., Jan. 22, 1900.

R. Deutsch[1] describes several cases which he considers acute gastro-intestinal autointoxication. In the first case there was a steady onset of status epilepticus in a girl of 15 who had previously been well. The child recovered entirely. Examination of the urine at the time of the attack showed **large amounts of acetone.** There was a history of dietetic indiscretion, and the case was considered to be gastro-intestinal autointoxication. In another case there were signs of central pneumonia and of meningitis in a 10-year-old child, the attack having come on very suddenly in the midst of good health. The urine in this case also showed large amounts of acetone, and the use of calomel caused recovery without any development of actual signs of pneumonia. The case was considered one of acetone intoxication. He also describes a case of intermittent ophthalmic neuralgia in which there was much indican in the urine, and in which the use of calomel caused complete recovery. In another case, with sciatica and erythematous eruptions associated with acetonuria, calomel caused rapid recovery.

Rendu[2] describes a case which he terms **alimentary intoxication of intermittent type.** The patient presented signs very similar to those of typhoid fever when he was admitted, except that there was marked disturbance of the stomach and an erythematous rash. The existence of these signs led to a diagnosis of gastro-intestinal intoxication, and this was thought to be confirmed by learning that the man had partaken of spoiled food. It was notable, however, that distinctly intermittent attacks of fever resembling tertian malaria appeared. These were controlled by quinin, and it is possible that the case was one of malaria the onset of which had been precipitated by the gastro-intestinal intoxication.

O. J. Kauffmann[3] describes cases in which there is, associated with **nervous disorders,** the appearance of **gastro-intestinal disturbance.** He says justly that there is some evident connection in certain of these cases between the gastro-intestinal disturbance and the progress or severity of the nervous disorder. In many of these he has found indican present in the urine, and speaks of them as instances of gastro-intestinal autointoxication, though other evidence of such a condition is absent from the report. [The term gastro-intestinal autointoxication is used with undue freedom. The mere fact that some other condition grows better or worse as gastro-intestinal disturbance grows better or worse does not indicate that the condition is one of gastro-intestinal autointoxication, nor does indicanuria. Undoubtedly gastro-intestinal autointoxication exists, but satisfactory knowledge of its true clinical features is not furthered by the careless use of the term.]

## DISORDERS OF THE URINE AND DISEASES OF THE KIDNEYS.

J. B. Nash[4] describes the results of his estimation of the urea and of the fluid excreted by the kidneys in 3 patients, 2 of whom had lost one kidney; the other had had about one-half of one kidney removed. The results of his work were the conclusions that 3 years after the removal of one kidney the remaining organ excretes more than the normal average amount of water, the average amount of urea being about normal. Two years after the removal of half of one kidney the urine was about normal in amount and the urea percentage the same. Immediately after the removal of the kidney in one case, and throughout a subsequent period of 7 weeks, the average daily excretion of water and urea was found lessened, but not below the amount commonly excreted by a person with normal kidneys who has been subjected to a serious operation.

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[1] Wien. med. Woch., Nos. 5 u. 6, 1900.

[2] Gaz. des Hôp., Dec. 22, 1899.

[3] Edinb. Med. Jour., April, 1900.

[4] Australas. M. Gaz., Dec. 20, 1899.

Widal,[1] in discussing the condition of the renal function in chronic nephritis, states that uremia may occur when the **permeability of the kidney to methylene-blue** seems entirely normal, even in early cases. The kidneys in the parenchymatous forms of nephritis do not show marked changes in the elimination of methylene-blue, but in the interstitial variety the elimination is greatly delayed. The differences in the elimination of methylene-blue are, however, not proportionate to the renal lesion. In discussion Archard stated that in investigating the permeability by the use of methylene-blue one should take into consideration both the time in which it is eliminated and the quantity which is eliminated. Often the elimination is protracted over a long period when the permeability is lessened, or the lessened amount given may be eliminated within 24 hours. Merklen considered the amount of urea eliminated a better index of the permeability of the kidneys than the permeability to methylene-blue.

Kloupet[2] discusses the **study of the functional capacity** of the kidney by the **use of phloridzin.** It is known that this substance causes glycosuria, and he considers that in these cases the glucose not only filters through the kidney, but is produced by the epithelium of the kidney. He thinks that it may, therefore, be used to test the functions of this organ. In chronic nephritis the glycosuria is reduced, and sometimes none occurs, or the elimination of glucose may be much prolonged. In acute nephritis, and in cases of fever with involvement of the kidney, the glycosuria is excessive.

E. C. Hill,[3] after examining over 6000 urines in Denver, gives the following statement concerning the **changes resulting from high altitudes:** Acute nephritis is not common, but when it occurs it is very severe; amyloid disease is not so common as text-books would indicate; chronic parenchymatous nephritis seems to run about the same course as in lower altitudes; and chronic interstitial forms run a more favorable course than in lower altitudes. Slight transient albuminuria is common in high altitudes.

C. W. Purdy[4] describes a method of determining the amount of chlorids, phosphates, and sulphates in the urine by **centrifugation.** He employs a centrifuge which has a double arm and carries 4 tubes. In each of 3 tubes he places 10 cc. of urine. To the first, for estimating chlorids, he adds 1 cc. of strong nitric acid and 4 cc. of standard silver nitrate solution. In the second tube, for the phosphate estimation, he places 2 cc. of 50% acetic acid and 3 cc. of uranium nitrate solution. To the third tube, to estimate the sulphates, he adds 5 cc. of standard barium chlorid solution. In the fourth tube he places 15 cc. of water to balance the centrifuge. The instrument is then revolved at the rate of 1200 revolutions a minute for exactly 3 minutes. The percentage bulk of precipitate is then read off from the scales on the tubes. Tables are given for determining the amount of salts present by means of this bulk percentage.

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[1] Semaine méd., Feb. 17, 1900.

[2] Gaz. hebdom. de méd. et de chir., Jan. 28, 1900.

[3] Jour. Am. Med. Assoc., May 12, 1900.

[4] Jour. Am. Med. Assoc., Feb. 10, 1900.

M. Cohn [1] describes the following method for the **preservation of urinary sediments:** The sediment is spread upon perfectly clean cover-glasses and allowed to dry in the air; the cover-glasses are then placed for about 10 minutes in a 10% formalin solution, being careful to avoid shaking; they are washed and then stained with a concentrated solution of Sudan in 70% alcohol, the staining lasting about 10 minutes; they are then washed in 70% alcohol, counterstained with alum-cochineal, and finally mounted in glycerin. A number of beautiful illustrations are given showing the value of this method.

L. N. Boston [2] recommends for the **preservation of urinary casts** a mixture of 1 ounce of the liquor of arsènious acid, ½ of a grain of salicylic acid, and 2 fluidrams of glycerin, which is warmed until solution occurs, and then saturated with "whole tears" of acacia. After the deposit has subsided the supernatant liquid is decanted; a drop of 40% formalin may be added. Some of the urinary deposit is placed upon the slide, and if casts are found to be present, it is evaporated nearly to dryness and a drop of the solution placed in the center of the drop of urine and gently mixed with a fine needle. A cover-glass is applied and the slide placed in a cool atmosphere for a few hours until it hardens. It is then surrounded by a ring of zinc white.

Biffi [3] describes the following **test for bile:** From 150 cc. to 200 cc. of urine is strongly acidified with sulphuric acid; then a 5% barium chlorid solution is added drop by drop, using about 30 drops to every 100 cc. of urine. The liquid is then poured off and the soft precipitate is collected on absorbent cotton and spread evenly over the surface. A crystal of potash bichromate is then placed upon the precipitate, and if bile is present, a green ring appears around the crystal, afterward changing to blue and then to red. A small amount of albumin does not interfere with the test; but if much is present, Biffi recommends the use of a saturated solution of sodium sulphate instead of the sulphuric acid.

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[1] Zeit. f. klin. Med., Bd. XXXVIII, Hefte 1, 2, u. 3.

[2] N. Y. Med. Jour., Nov. 4, 1899.

[3] Gaz. degli Osped., Feb. 11, 1900.

## DISORDERS OF THE URINE.

Cammidge and Garrod [1] describe a case of **cystinuria** in a man of 22 in which they were able to demonstrate the presence of **diamins** in the urine and feces. They found that the excretion of diamins was extremely intermittent, and that these bodies disappeared from the urine for as long as 23 successive days. They decided, therefore, that systematic examination of the excreta over protracted periods may show that diamins are more frequently present in the urine in cystinuria and other conditions than is commonly taught.

C. E. Simon [2] discusses cystinuria, and describes a case in which diamins were also found in the urine. Cystinuria is apparently due to **insufficient nitrogen metabolism,** and seems to belong in the same category of diseases as diabetes, gout, obesity, and the like. Simon believes that cystin is derived from the nonhydrated benzyl radicle of the albumins. He reports a collection of 103 cases of cystinuria.

**Bacteriuria.**—Warburg [3] describes a case of bacteriuria in a man of 54 who had previously never had any disease of the genito-urinary tract. The attack came on with chill and fever, the only other sign being turbidity of the urine, which proved to be due to the presence of a small bacillus in large numbers. There were no casts or pus-cells. The fever disappeared by the fourth day, but the bacilli were still present in quantities. Salol was ineffective, but urotropin caused the urine to clear up almost immediately. The portal of entrance of the bacillus was not determined. The organism proved to be **Bacillus lactis aerogenes.**

**Proteids of the Urine.**—W. B. Hills [4] has studied the **proteids of the urine** in 73 cases of acute and chronic nephritis, secondary congestion of the kidneys, and inflammation of the urinary mucous membranes. From his study of these cases and of the literature Hills decides that urines containing albumin show globulin also in most cases, and that globulin is present in a certain number of cases as the only coagulable proteid. In some cases albumin is also probably the only coagulable proteid to be found. The significance of globulin is uncertain, but it is probable that an increase of the proteid quotient—*i. e.*, the number obtained by dividing the quantity of albumin excreted in 24 hours by the quantity of globulin—is of favorable prognosis, while a decrease in the quotient is unfavorable.

**Albuminuria.**—H. P. Hawkins [5] discusses **albuminuria in the apparently healthy** under the forms of exertion albuminuria and neurotic, dietetic, and postural albuminuria. The evidence which has been adduced tends to show that the trouble is situated in the vasomotor apparatus which regulates the blood-flow into and from the glomeruli, and that there is no disturbance of the actual kidney structures. The prognosis he considers good, but the patient should lead a careful life, and the urine should be examined repeatedly every year. In the forms due to exertion, diet, or cold baths the removal of the cause will usually end the trouble, but in the other forms active treatment is indicated.

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[1] Jour of Path. and Bacteriol., Feb., 1900.

[2] Am. Jour. Med. Sci., Jan., 1900.

[3] Münch. med. Woch., July 18, 1899.

[4] Boston M. and S. Jour., Aug. 10, 1899.

[5] Brit. Med. Jour., Dec. 9, 1899.

Rudolph,[1] in discussing **cyclic albuminuria,** states that he believes that it is, as Stirling thinks, largely the result of the assumption of the upright posture upon arising from bed. He believes, however, that it is not due to the shock experienced by the kidneys from the pressure of the column of blood above them when the position is changed from the horizontal to the upright, but is merely the result of the change in blood pressure from the change in position without the action of any shock. He thinks that the affection is an albuminuria of venous stagnation, and that it is the result of previous inflammation. The change in the kidneys has been only slight, and such as to cause some loss of elasticity in the vessel-walls of the glomeruli, so that with increased pressure they more readily allow the albumin to pass into the urine. He considers such patients always ill, however. He does not believe that they should be kept in bed, as the affection is rarely cured in this way, and the general condition is likely to suffer. The most satisfactory treatment is that directed to improvement of the general condition.

**Albumosuria.**—L. L. Aldor [2] discusses albumosuria, and particularly the **enterogenetic form.** The tests for albumose have all had fallacies, and urobilin especially responds freely to the usual tests. Aldor reports a method which he thinks eliminates the error caused by urobilin. He puts from 5 cc. to 10 cc. of urine in a test-tube and adds 1 or 2 drops of hydrochloric acid, and then phosphotungstic acid until no precipitate occurs. He then centrifugates, pours off the fluid, shakes the sediment with alcohol, and pours off the alcohol, repeating the process 2 or 3 times and then adding concentrated sodium hydrate to the sediment. A blue color appears, which vanishes after shaking in the air. The biuret test is then applied. He investigated 53 cases of various kinds, and a connection was observed between fever and albumosuria, albumose being found in about 90% of febrile cases. It was observed in about 4 cases of carcinoma of the stomach and in 1 of peritoneal carcinoma ; in these cases he does not consider the albumose as of intestinal origin, and does not think that one can draw any conclusion concerning the seat of carcinoma from the presence or absence of albumosuria. The presence of albumose in the urine indicates abnormality of metabolism, but no definite diagnostic conclusions can be drawn from its presence or absence.

**Nephritis.**—P. F. Richter and W. Roth [3] present an experimental **study of kidney insufficiency,** the method of study being the determination of the freezing-point under various conditions. Their results were that in the rabbit's blood the depression of the freezing-point is about normal, being about 0.56. Double nephrectomy causes increase of the molecular concentration of the blood by accumulation of metabolic products. The removal of one kidney does not have this effect, because the remaining kidney compensates for the lack of the other; but if the kidney that is left is injured, the molecular concentration becomes excessive. The freezing-point is depressed pathologically in cases of bilateral nephritis resulting from poisoning. There is a molecular retention in nephritis which is more marked when the disease is diffuse, and particularly affects the vascular apparatus; when the tubules are chiefly affected, the retention is not so pronounced. Artificial infarction of the tubules leads to retention in the blood, and pathologic depression of the freezing-point is the result of the retention of metabolic products, and not merely of sodium chlorid. In some cases the pronounced anemia and dropsy interfere with the pathologic depression of the freezing-point. A study of the concentration of the blood will be chiefly of value to surgery, where it is often extremely important to determine the functionating power of the kidneys before operation on these organs is carried out.

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[1] Centralbl. f. innere Med., Mar. 3, 1900.

[2] Berl. klin. Woch., Sept. 4, 1899.

[3] Berl. klin. Woch., July 24, 1899.

Koranyi [1] reports further work which he has carried on concerning renal insufficiency, using the degree of reduction of the freezing-point in order to determine the concentration of the urine and blood. As conclusions he states that the **metabolism of the proteids produces the poison of uremia.** Increase in osmotic pressure occurs in renal insufficiency, and is due to the retention of the products of albumin metabolism; this is an important factor in the production of dropsy, which is due to the retention of solid molecules and the consequent retention of water. In cases of insufficiency of the kidneys, therefore, albuminous metabolism should be limited, which limitation may be effected by a suitable diet or, better, by the **use of curare.**

Lindemann [2] discusses the **concentration of the urine and of the blood** in disease of the kidneys and in uremia. His method was the determination of the nitrogen, chlorids, and freezing-point, as well as the total amount of urine and the specific gravity. He investigated normal urine and urine in various forms of nephritis. The freezing-point in normal urine usually averaged between 1.3° C. and 2.3° C.; more rarely normal urines varied within wider limits. The concentration is much lessened in interstitial nephritis, not so decidedly lessened in the parenchymatous form. In the interstitial form the concentration may be less than that of normal blood-serum. No absolute distinction can be made in this way between acute and chronic nephritis, but it is a valuable method, Lindemann thinks, of determining the degree of improvement in the kidney, or, on the other hand, of tendency to contraction. In forms of albuminuria not associated with nephritis there is no diminution in the depression of the freezing-point, hence one may determine whether the nephritis is present or absent, and one may also determine the occurrence of nephritis complicating cystitis or pyelitis. In uremia the freezing-point of the blood was much lowered. This result seemed to be due to a diminution of the amount of water in the tissues; the same condition can be produced by withholding water or by injecting strong solutions of sodium chlorid.

[1] Berl. klin. Woch., Sept. 4, 1899. [2] Deut. Arch. f. klin. Med., Sept. 29, 1899.

G. Kövesi and W. Roth-Schulz[1] discuss the **disturbances of the water-secreting activity** of diffusely diseased kidneys. In cases of advanced nephritis, when compensation is no longer possible, the freezing-point is abnormally high; there is, in other words, a molecular oliguria. Under normal conditions the kidneys have a marked power of accommodating themselves to circumstances in eliminating different amounts of water. In parenchymatous nephritis much of this power of secreting dilute urine is lost, while in interstitial nephritis it is not involved to any extent. In heart disease, if the compensation is lost, the diluting power of the kidney is diminished, while in compensated heart lesions it is not involved to any extent. The determination of the freezing-point and of the quantity of urine excreted in a day shows whether there is molecular oliguria, and the determination of the freezing-point of the blood shows whether the kidneys are removing waste products properly.

A. R. Elliott[2] insists upon the **frequency of granular kidney,** and in noting the fact that hemorrhages may usher in the symptoms of this disease, states that in 3 recent instances which he has observed the first symptom was in one case hemoptysis, in another epistaxis, and in the third severe renal hematuria. He insists upon the importance of heredity in the etiology of the disease, and believes that such a history renders the prognosis much graver.

P. K. Pel[3] records the histories of a series of cases illustrating the **family tendency to chronic nephritis.** In one family in 3 generations there were no less than 18 cases of chronic nephritis, in all instances appearing when the subjects were moderately or well advanced in years. All the subjects finally became uremic and died in coma. A curious fact noticed was that in the third generation the sons of nephritic fathers acquired nephritis, but the daughters did not; but when the mother had nephritis, the daughters acquired the disease, but the sons did not. Another noteworthy fact was that one small child whose mother had nephritis died of uremia with acute nephritis as a complication of chicken-pox, and since this mild affection is rarely accompanied by kidney trouble, it seemed to indicate that there was an especial hereditary predisposition to nephritis in this child also. The remainder of the fourth generation is still too young to state whether the predisposition has been further transmitted. Pel believes that there was in these cases a definite transmission of a tendency to disease of the kidneys.

H. Eichhorst[4] reports a case of fatal acute nephritis in a child of 12 which **resulted from eating sorrel** in large amounts. The case is noteworthy from the fact that children are so likely to eat this plant in their play, and serious poisoning is an extremely rare result; probably this is the only case as yet recorded in which the poisoning was fatal. The oxalic acid contained is undoubtedly the cause of the intoxication.

L. Lewin[5] believes that Eichhorst's report of death from nephritis as a result of poisoning by sorrel was based upon insufficient grounds.

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[1] Berl. klin. Woch., April 9, 1900.

[2] Med. Rec., July 15, 1899.

[3] Zeit. f. klin. Med., Bd. XXXVIII, Hefte 1, 2, u. 3.

[4] Deut. med. Woch., July 13, 1899.

[5] Deut. med. Woch., July 27, 1899.

Eichhorst offered no proof that other plants were not eaten with the sorrel, and Lewin thinks that there is no proof that sorrel is ever a dangerous poison.

Lafforcade[1] investigated **the edematous fluid** of Bright's disease, especially as to its toxicity. He found that it caused death only when given in very large doses—250 cc. per kilogram of body-weight. This is less toxic than normal urine or even blood-serum, and the only symptoms produced were similar to those caused by the injection of artificial serum. This shows that the poisons causing uremia do not accumulate in serous exudates.

Köhler[2] investigated the amount of **nitrogen excreted in the sweat** in renal disease. There has been much difference of opinion concerning this question. He decided from his investigations that no definite increase in the quantity of nitrogen in nephritis in general could be determined, but that marked increase of perspiration increased the excretion of nitrogen through the sweat. He found in one case, however, that although there was a marked increase in the quantity excreted in the sweat, uremia occurred the next day; therefore he concluded that sweating did not necessarily prevent uremia.

R. C. Cabot and F. W. White[3] have so far as possible studied all the cases of chronic nephritis which have been treated in the Massachusetts General Hospital, following the histories of patients either by communicating directly with them or with their physicians. The purpose of the study was chiefly to determine **whether chronic nephritis is curable** or not. They decide that recovery does occur in rare instances, and that the disease may exist for a long term of years without causing any evident constitutional disturbance. The average duration of life in 322 cases of chronic nephritis was found to be 19 months. They believe that acute nephritis is much less common than is usually thought, and that many cases so considered are really instances of exacerbation of chronic nephritis.

C. O'Donovan[4] contributes a paper on the **importance of Cheyne-Stokes respiration** during sleep as an early indication of interstitial nephritis, recording several cases. The first was that of a man of 54, whose previous history was without important incidents. He showed no physical signs of disease and the urine was negative, but, especially after excitement or following great muscular exertion, he had for 7 years frequent attacks of Cheyne-Stokes respiration at night. A few months before his death the diagnosis of Bright's disease was made from the urinary and general signs, and he died of renal insufficiency. During the latter part of his life the Cheyne-Stokes respiration was noticed in the daytime. In another case the Cheyne-Stokes respiration appeared 4 years before death, and occurred almost entirely at night.

Labadie-Lagrave, Boix, and Nové[5] report that they have determined that there is no relation between the albumin in the urine and the coefficient of the urinary toxicity either in Bright's disease or in other affections. They believe that the **gravity of a case** of Bright's disease should be determined by investigating the **toxicity of the urine** rather than the amount of albumin present in the urine.

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[1] Gaz. hebdom. de méd. et de chir., Jan. 28, 1900.

[2] Deut. Arch. f. klin. Med., Feb. 6, 1900.

[3] Boston M. and S. Jour., Aug. 10, 1899.

[4] Med. News, Sept. 16, 1899.

[5] Compt. rend. de la Soc. de Biol., Feb. 7, 1900.

T. R. Brown,[1] in discussing some **urinary anomalies,** reports cases of what he terms **simulative nephritis.** The first case was observed after an operation for floating kidney, in which an exploratory incision was made in the kidney and afterward closed, and the kidney stitched to the posterior abdominal wall. The operation was followed by the appearance in the urine of numerous blood-cells and epithelial cells and of enormous numbers of casts. Investigation of the reports showed other cases which resembled this. In 2 of them the urinary changes were very marked. In 3 cases simple microscopic examination of the urine after the operation alone would have led to a diagnosis of acute nephritis; albumin, however, was present only in small amounts, and there were none of the clinical signs and symptoms of nephritis. The condition was transitory. It was attributed to the handling and stitching of the kidney, the irritation caused by the ether, and the diminution of the fluids which occurs after ether anesthesia.

V. Curcy,[2] in making a microscopic examination of the sediments of the urine from a case of severe genito-urinary infection, which readily gave way to the use of balsams and antiseptic washes, found large numbers of **Tetragenus septicus;** the micro-organism was very virulent when injected into animals.

**Complications.**—A. D. H. Leadman [3] reports the case of a man of 46 who had severe Bright's disease, and was suddenly taken with pain and helplessness in both legs, the latter becoming cold. **Gangrene** appeared in the feet and above both knees the next day. Neither femoral artery pulsated. The gangrene advanced continuously until the patient died, about a month later, and at death both lower extremities were completely involved. The diagnosis of the case was occlusion of the abdominal aorta. No postmortem could be obtained.

De Fleury [4] reports 3 cases of Bright's disease in which there occurred **nervous disturbances:** in one, a right-sided transitory hemiparesis; in the second, melancholia with delusions of persecution; and in the third, attacks of petit mal. The nervous symptoms disappeared entirely after the use of a milk diet.

C. Gerhardt [5] has observed **blindness for blue** in a number of cases of cirrhotic kidney—a condition which Koenig had previously described.

F. Krauss [6] describes a case of uremia in which there were severe convulsions with **conjugate deviation** and **hemiplegia of the opposite side.** The urine contained no albumin. Postmortem examination showed no lesion of the brain except congestion and edema.

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[1] Johns Hopkins Hosp. Bull., May, 1900.

[2] Centro farmaceutico urogayo, July 15, 1899.

[3] Brit. Med. Jour., April 14, 1900.

[4] Bull. Acad. de méd., Oct. 31, 1899.

[5] Münch. med. Woch., Jan. 2, 1900.

[6] Phila. Med. Jour., April 14, 1900.

**Treatment (Diet).**—Von Noorden [1] condemns the time-honored method of prohibiting **red meats** in dieting cases of chronic nephritis. One common result, he states, is that patients who are forbidden the use of red meats grow so disgusted with white meats that they take too little albumin; if they are allowed red meats, they at once improve and gain strength. He has found no reason to believe that there is any exact basis for the statements that have been made that the red meats are more irritating to the kidneys. Von Noorden states that the amount of extractives in red meats, contrary to the usual teaching, is not greater than that in white meats, the real difference being in the amount of coloring-matter. He tested one patient with chronic Bright's disease by giving him for one period white meat of poultry, and for the next period an equivalent amount of nitrogen in beef. The result was that he excreted the same amount of nitrogen in both periods, and a little more albumin in the first period than in the second. Von Noorden also believes that in many cases much good may be done by restricting the fluids to between 40 and 50 ounces, especially when there is dilation of the heart and a tendency to cardiac asthma. The cardiac area diminishes, the asthmatic attacks grow less frequent, and the patients sleep better, and the restriction does not interfere with proper alimentation.

Offer and Rosenquist [2] have investigated the correctness of the teaching that **white and dark meats** differ in the amounts of extractives which they contain. They demonstrate that the earlier investigations have been fallacious, and as a result of their determinations conclude that, while at times white meats do contain less nitrogen and other extractives, as a rule there is very little difference in the two forms of meat, and there is no rational basis for using white meats exclusively in cases in which it is desired to limit the amount of extractives. The only method of doing this is to limit the amount of meats.

H. A. Hare [3] believes that patients with contracted kidney, so long as they are not decidedly gouty subjects, may be allowed a fairly liberal diet if it does not disturb digestion. He does not cut off meats to any extent, allowing even red meats and eggs. In acute nephritis he advises the avoidance of albuminous foods during the active course of the disease. If oliguria depends upon the renal inactivity, he administers caffein and potassium nitrate in infusion of juniper; if the heart is feeble, he gives cardiac stimulants. Water should not be given in large amounts if there is dropsy already; otherwise it may do good. He considers that iron is relied upon too much, and is likely to be given in too large doses. He does not recommend the use of pilocarpin or other medicinal diaphoretics. Pilocarpin is dangerous because of its depressing effects, and because of its causing too free excretion into the bronchial tubes. If it is given, strychnin should be given with it.

W. Leube [4] gives a highly interesting discussion concerning the **compensatory changes in disease,** dividing them into: (1) Those in which a part of the organ becomes diseased and the remainder compensates, or in which one of a pair of organs becomes diseased and the remaining one compensates; (2) those cases in which certain organs take up the function of diseased organs, the two classes having about the same general function—as, for instance, when the skin compensates for disturbances in the kidneys; (3) instances in which organs normally not having a function similar to the diseased organs assume to some extent the functions of these organs. Under the last head he describes instances in which an excessively large secretion of saliva resulted in the absorption of exudates. In one instance the salivation occurred spontaneously, the man secreting as much as **3 liters of saliva** a day, with the result that a large ascites entirely disappeared. This happened repeatedly, and at times the saliva was almost spurted out of the mouth in attacks of excessive secretion. The postmortem showed that cirrhosis of the liver was present. In order to make use of this observation Leube repeatedly had patients with collections of fluid chew tablets of rubber, and in this way he was easily able to cause a secretion of from 400 cc. to 1000 cc. of watery saliva; in 5 cases of pleural effusions he was able in 4 instances to cause complete absorption of the exudate in a short time, while in 2 cases of ascites the result was negative in one and completely satisfactory in the other. He thinks that excitation of salivation may come to be a method by which we can replace diuresis and diaphoresis if there be necessity for this. He recommends that combined salivation and diaphoresis by means of pilocarpin be tried in these cases. Under the heading of the vicarious action of organs whose functions are related to each other he discusses the value of diaphoresis in diseases of the kidneys. If there is marked edema of the skin, it may repeatedly be observed that free sweating may be produced without the excretion in the sweat of much organic material; the result is that while the subcutaneous collections of fluid originally contained a good deal of excrementitious material, the loss of water increases the concentration of the blood, and the result of this is the absorption from the subcutaneous tissues of poisonous material. Leube and others have often noticed in cases of nephritis with severe general anasarca that **uremic attacks** came on **after severe sweats,** evidently owing to the absorption in the manner described. Hence he insists that sweating should be used with the greatest care in cases of nephritic anasarca; but the question is an entirely different one when there is nephritis without any marked skin edema. He does not consider that there is any testimony that the stomach or intestines have marked vicarious action in kidney disease; there is no good evidence that urea is at all constantly or freely excreted by the gastro-intestinal tract in nephritis, even when the functions of the gastro-intestinal tract are excited. As to the vicarious functionation of blood-producing organs, he notes that in 434 cases of chlorosis which he has seen, no less than 71 showed spleens which were distinctly palpable. He considers that this is unquestionably a compensatory enlargement of the spleen. Whether the spleen in such cases acts by actually producing blood-cells or by preparing material for new blood-cells from those that are destroyed is uncertain. In such cases, however, he has had better results from the use of quinin and iron together than from iron alone.

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[1] Verhandl. d. Cong. f. innere Med., 1899.

[2] Berl. klin. Woch., Oct. 23 and 30, 1899.

[3] Therap. Gaz., Dec. 15, 1899.

[4] Deut. Arch. f. klin. Med., vol. LXVI, 1899, Festschrift.

P. F. Richter[1] has attempted to discover the nature of the **favorable influence of venesection** on uremia. He determined the molecular concentration of the blood before and after venesection, both with and without saline infusion, but found no diminution in the molecular concentration or in the osmotic pressure. Hence a change in the molecular concentration of the blood can not be advanced as an explanation of the results.

J. B. Walker[2] believes that in the **climatic treatment** of chronic interstitial nephritis it is quite as important to look to the nature of the soil as to atmospheric conditions, and he thinks a dry sandy soil with an equable climate, such as those of the New Jersey coast, to be quite as valuable in treatment as places in the West or South.

**Ureteritis.**—C. G. Stockton[3] in discussing spurious albuminuria, states that there has not been sufficient attention given to the fact that traces of albumin are found in ureteritis, a condition which he considers is likely to be mistaken for appendicitis or disease of the uterine adnexa. In two cases he observed the affection in women after parturition; both had floating kidneys, and it was considered probable that the ureteritis was the result of transient hydronephrosis caused by pressure of the fetal head on the right ureter. In both cases there was high temperature, and the urine contained a little albumin with some pus and blood-corpuscles. Both patients recovered entirely.

**Movable Kidney.**—A. Stengel[4] describes a belt which he has had made for the treatment of movable kidney. He believes that the older belts are often useless or worse than useless, because the pressure is directed in an improper way. The shape of the kidney and its manner of attachment are such that it does not move directly upward and downward, but from the right above to the left below; hence Stengel had a **special pad** with a convex upper surface, the shape being roughly reniform. This is covered with leather and made soft though firm. It is about 3 inches long and about 2½ inches wide. It is to be held in place by an abdominal belt of knitted silk or cloth with perineal straps, and the pad so placed that the concave border faces upward and toward the right and lies a little below the line passing from the umbilicus to the anterior superior spine of the ileum; it should look upward toward the tip of the eighth rib. A pad of this form will press the kidney back into its normal position, and has given great relief in 4 cases in which Stengel has used it.

**Cystic Degeneration.**—Steiner[5] records a case of cystic degeneration of the kidneys and liver in a man of 52. The diagnosis had been made during life, owing to the association of bilateral nodular tumors, which appeared to be of the kidney, and an enlargement of the liver, which showed the same hard and painless nodules. The postmortem examination confirmed the diagnosis. Another case came to his notice soon afterward, and this led him to investigate the family history of such patients. He found that the first patient had a son and a sister who had cystic kidneys, and that in several of the patient's younger children there were suspicious nodules on the surface of the kidneys. In the family of the second patient it was noticed that a brother and a sister, the only other members of the family in the generation of the patient, had cystic kidneys; a 10-years-old son of the brother also showed the affection and a number of children in his family showed albuminuria. Steiner decides, therefore, that the disease is notably hereditary in character. Enlargement of the kidney does not begin on both sides coincidently, but is seen much earlier on one side. This is of importance, since it is very likely to lead to a false diagnosis of malignant tumor. The association with cysts of the liver is believed to be of marked importance in establishing the **diagnosis.** The treatment is practically negative. Operation is scarcely to be considered in these cases, as the patients are usually weakly and there is great danger of production of a fistula, and the effect upon the course of the disease is usually negative.

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[1] Berl. klin. Woch., Feb. 12, 1900.

[2] Med. News, Sept. 16, 1899.

[3] Jour. Am. Med. Assoc., Sept. 28, 1899.

[4] Univ. Med. Mag., Sept., 1899.

[5] Deut. med. Woch., Oct. 12, 1899.

**Tuberculosis.**—D. Newman [1] discusses the occurrence of **hematuria** in its relation to tuberculosis of the kidney. He particularly notes that the first bleeding may occur from injury, but that the point of hemorrhage and the surrounding injured tissues may become tuberculous as a secondary result of the injury. He describes two cases of this kind, in both of which there was severe injury in the region of the kidney, with hematuria; in one after 6 months, and in the other after 2½ years, symptoms of tuberculosis of the kidney, with bacilli, blood, and pus in the urine, developed. He notes, however, that tubercle bacilli may be excreted by the urine, and that their presence therein is not a pathognomonic sign of tuberculosis of the kidney. He also insists that the hematuria may be a premonitory sign of tuberculosis of the kidney, and that the actual disease may show itself only when years have passed subsequent to the hematuria. A case of this kind is noted in which the tubercular disease seemed to appear 2 years after hematuria; in another case a man had had marked hematuria 13 years before distinct signs of tuberculous pyonephrosis developed.

J. Frank [2] describes the case of a woman of 39 who had manifested symptoms diagnosed as cystitis for 20 years. They consisted chiefly of dysuria, pyuria, and polyuria, with a burning pain in the back. At times there were symptoms similar to those of renal colic. Catheterization of the ureters showed that the urine from the left kidney was normal, while that from the right contained pus, blood, and tubercle bacilli. The right kidney was removed by lumbar nephrectomy, and the patient apparently recovered entire health.

J. M. Da Costa [3] describes the case of a man of 23 whose symptoms began with severe pain in the lumbar region transmitted to the right scapula. When first admitted to the hospital, there was slight albuminuria, but there were no other abnormalities in the urine. The pain disappeared completely in 2 weeks; the albumin also vanished, and the man's condition improved greatly. He had no other evidence of tuberculosis except a disposition to take cold, but repeated examination of the urine showed the presence of bacilli, which showed the characteristic staining of tubercle bacilli. A diagnosis of **tuberculosis** of the kidney **without general symptoms** was made. [It must be remembered that a diagnosis based upon the discovery in the urine of bacilli which resemble tubercle bacilli is always subject to error unless great care has been taken to exclude the presence of the smegma bacillus.]

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[1] Lancet, Aug. 26, 1899.

[2] Med. Rec., Sept. 9, 1899.

[3] Phila. Med. Jour., Jan. 20, 1900.

**Anomalies of the Kidney.**—D'Arcy Power[1] describes the case of a man of 36 who had suffered from almost complete **suppression of urine** for a week. He had manifested no previous signs of renal calculus, and operation upon the right kidney disclosed no stone. The man died. The next day at autopsy the left kidney was found to consist only of a small mass of fibrous tissue without any renal vessels. In the pelvis and remaining kidney there were 4 small calculi, and death was thought to be due to calculous suppression of urine.

O. Buss[2] records the case of a woman of 21 on whom a laparotomy was performed with the intention of removing the ovaries in order to do away with menstruation. There was absence of the vagina and she still menstruated, and a tumor of considerable size had formed which was believed to be a hematometra. The left ovary was found and removed, but the right could not be found. The tumor also was removed, and was found to be a dislocated kidney, which showed marked fetal lobulation and was severely diseased, being hypertrophic in some areas and atrophic in others. The chief interest in the case was that dissection showed the absence of any appearance of a uterus except a flat band of tissue containing a few muscular fibers, and the right ovary was absent. The right kidney was also found to be entirely absent, and yet in spite of the **complete absence of any secretion of urine** after the operation, the patient had lived 7 days. The temperature became subnormal and there was an appearance of uremia with vomiting and eructation, followed by contraction of the pupils, restlessness, and marked atrophy. There were no convulsions, the only suggestion of their occurrence being clonic contractions of the lower lip. There was also no vomiting. This duration of life is much longer than any previously reported under similar conditions.

## PARASITES.

E. Walker[3] describes a case of **Bilharzia hæmatobia** in a woman of 28. She had been operated upon for ovarian cyst, and subsequently had pain in the region of the bladder and repeated hemorrhages. The cyst on the other side was then removed. After this she had, however, return of the pain and hemorrhage, frequent abdominal distention, and often a sudden passage of a considerable quantity of fluid of very low specific gravity, from which urea was almost entirely absent. Gas was sometimes discharged with the fluid from the bladder. The fluid contained ova and embryos of Bilharzia, and two young worms were discovered.

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[1] Lancet, Jan. 6, 1900.

[2] Zeit. f. klin. Med., Bd. XXXVIII, Hefte 4, 5, u. 6.

[3] Jour. Am. Med. Assoc., Feb. 17, 1900.

C. P. Childe [1] records the case of a girl of 16 who had lived in Natal and who presented **hematuria and irritability of the bladder** and showed small particles floating in the urine. The examination of these particles showed the presence of Bilharzia. The infection was believed to have occurred through bathing in a fresh-water pool.

J. Gutsch [2] describes a case of hematuria resulting from Bilharzia infection which occurred in a boy of 17 who until 2 years previously had been in South Africa. He was probably infected through bathing or drinking water from streams. He soon disappeared from observation.

F. M. Landwaith [3] found **Distoma heterophyes** in the feces of a patient who was also the subject of **Bilharzia** of the bladder and rectum. The distoma was found several times, after which it disappeared, but the ova were seen for several weeks subsequently. They also disappeared after that period, and the disappearance of the parasites was believed to be due to the use of a dose of 30 grains of thymol, which was given because of the Bilharzia affection. The latter parasites were not affected by the thymol.

H. Innes [4] describes the case of a boy who had pain in the right knee-joint, from which he observed a portion of a worm protruding. An incision was made and a piece of **guinea-worm** about 4 inches in length was removed. After drainage of the joint entire recovery occurred.

J. Patterson [5] describes the case of a man of 29 who was under treatment for an old sinus in the left calf, which was cured. Later, however, there was a swelling upon the inner malleolus on the same side, which was incised, and from the abscess cavity there was extracted a worm 5 inches in length, which proved to be the dracunculus. Some time before, he had had an abscess over the left tibia, and from this, it was said, a worm 4 inches long had escaped. It was possible that he had been infected while on the Gold Coast some years before.

E. Hawkins [6] reports the case of an **adult infected** with **Oxyuris vermicularis.** For over 12 years various forms of treatment had been ineffective, but the worms finally disappeared entirely after the man had been given regular doses of cascara.

R. W. Gray [7] reports the discovery of **Ankylostomum duodenale in the intestines of 3 dogs.** He finds these parasites common in the tea-garden collies of Assam, and since pigs and cows often eat fecal matter, it is likely that these animals become infected in this way.

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[1] Brit. Med. Jour., Sept. 9, 1899.

[2] Brit. Med. Jour., May 19, 1900.

[3] Lancet, Sept. 30, 1899.

[4] Brit. Med. Jour., Feb. 3, 1900.

[5] Med. Rec., Oct. 7, 1899.

[6] Brit. Med. Jour., Dec. 23, 1899.

[7] Brit. Med. Jour., Oct. 21, 1899.

**Filariasis.**—L. Rénon [1] reported the case of a man from Guadeloupe who had filarial infection, which showed itself practically solely by the occurrence of **testicular crises**; they had lasted for 10 years and came on without prodromes in the form of excruciating pain in the right testicle. This organ immediately became large and tender, and the pain radiated to the right iliac fossa and showed considerable resemblance to renal colic. The attacks were sometimes accompanied by fever. The blood contained embryos of filaria. He had had transitory edema of the scrotum and of the right foot, but no other edema. The difficulty in diagnosis was chiefly from renal colic. Filaria have been found repeatedly in the lymphatic tissue of the testicle and probably caused the symptoms in this case by their local effects.

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[1] Gaz. des Hôp , Mar. 16, 1900.

# PEDIATRICS.

By LOUIS STARR, M.D., and ALFRED HAND, Jr., M.D.,
OF PHILADELPHIA.

## GENERAL CONSIDERATIONS.

**Morbidity.**—T. Escherich, [1] after studies of the morbidity of children in the different age-classes, remarks that the morbidity, greatest in the first year, sinks rapidly, and shows a change in character from one age-group to another according to the diseases prevalent in that age; this change is due, on the one hand, to physiologic characteristics of the organism, and, on the other, to the external conditions and habits of children, which for the individual of one age-group are much more similar than is the case with adults. In the period of early infancy is shown the immature state of the organs, in functional weaknesses, especially of the digestive organs, and in a lack of adaptability to the circumstances of extra-uterine life; the skin and mucous membranes form a feeble protection against bacterial invasion and toxins, leading, therefore, to sepsis in the new-born and to infectious inflammations of the mucous membranes in nurslings. During the last half of the first year appear those constitutional diseases which are characterized by a disturbance in the growth of the organism, such as rickets, anemia, scrofula, which reach their height in the second or third year of life and form the main clinical picture of this period, with an association of so-called "filth-infections," due to the unclean habits of children of this age, and comprising diphtheria, pertussis, local tuberculosis, stomatitis, helminthiasis, etc. In the school period, from the fifth year on, the acute exanthemata reign, with their effects on the heart and kidneys; functional disturbances due to schooling—the school diseases—appear, analogous to the occupation diseases of adults. From this time on, the diseases resemble more and more those of adults—typhoid fever, pneumonia, etc. From the period of birth, syphilis shows a rapidly diminishing, tuberculosis a steadily increasing, frequency; the latter tends to the localized form, especially in the lymph-glands, and this leads to a special type of lung infection, miliary tuberculosis being the most frequent form up to the fourth year. From the standpoint of the physician, childhood may be regarded as a period of preparation, during which there develop, on the one hand, wonderful adaptability to the most varied climates and conditions of life, and, on the other, a vital resistance to infection in order that in adult life the struggle for existence may be survived with success. It is the physician's duty to watch over this process of acclimatization, striving to keep the harmful agencies from overbalancing the strength.

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[1] Jahrb. f. Kinderh., Bd. LI, Heft 1.

**Uric Acid.**—F. Goeppert[1] gives the result of researches on the elimination of uric acid, with a review of the literature. There is practically a constant relation between the elimination of uric acid and of total nitrogen on a mixed diet; but certain substances, such as the thymus of the calf and other nuclein-rich tissues, have a uric-acid-forming power greater than their proportion of N would indicate. On the other hand, a diet of eggs, perhaps milk also, and a number of proteid foods, forms relatively less uric acid than a mixed diet of equal N content. The nonnitrogenous foods, fat, carbohydrates, alcohol, and water influence proteid metabolism, but do not alter the factor of the total N to the uric acid N.

J. Comby[2] treats of **uricemia in children,** referring at length to Rachford's investigations on xanthin, paraxanthin, and heteroxanthin. Cases are briefly detailed showing the different manifestations of lithemia, and a didactic summary is given, stress being laid, in the etiology, on heredity. The symptoms are manifold, but may be grouped according to the system affected, thus: (1) Nervous symptoms, headache (paroxysmal or periodic), neuralgic pains in the bones or joints, cerebral excitement, insomnia, convulsions, neurasthenia; (2) digestive troubles, cyclic vomiting, colic, polyphagia, constipation, membranous enteritis, and intestinal lithiasis; (3) urinary conditions, renal calculus and colic, albuminuria, glycosuria, dysuria, and spasm of the bladder; (4) in the respiratory tract, coryza, sneezing, laryngitis, bronchitis, asthma, pulmonary congestion; (5) in the circulation, palpitation, tachycardia, arhythmia, false hypertrophy; (6) in the skin, eczema, sweating, pruriginous eruptions. In the treatment, a paragraph full of suggestions is devoted to the subject of mineral waters appropriate to the different conditions. For the joint pains, Bourbonne (Haute Marne) is recommended; for abdominal pains with diarrhea, Plombières (Vosges); for constipation and membranous enteritis, Châtel-Guyon (Puy-de-Dôme); for albuminuria, St. Nectaire (Puy-de-Dôme); for renal gravel, Contrexéville (Vosges); for gouty anemia, Royat, La Bourboule (Auvergne), St. Gervais (Savoie); for the dermatoses, Uriage (Dauphiné), Luchon (Haute Garonne), La Bourboule; for neurasthenia, Bagnères-de-Bigorre (Pyrénées); for dyspepsia, Vichy (Allier), Pougues (Nièvre).

**Milk Diet.**—M. Hoefler[3] gives a number of practical points with reference to the adoption of a milk diet, his remarks bearing only on its use for older children and not for infants. He states that when it is necessary to give a course of iodin or mercury, large amounts of milk, practically a milk diet, will make the effects appear more promptly and be more permanent, the combination between the mercury or iodin and the albumin of the milk being specially adapted to combat certain other toxic substances in the system. When patients object to taking milk, and say they can not digest it, the reason is because they are used to a diet which irritates the stomach and prevents it from handling the milk; and when this is corrected, and the milk given under certain regulations (warm and when the stomach is empty), children almost always develop an appetite for it; if constipation arises, salt may be added. The regulation of the diet so that milk may be taken will include abstention from beer, wine, fruit, salads with vinegar, and eggs and milk cooked together.

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[1] Jahrb. f. Kinderh., Bd. LI, Heft 3.

[2] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.

[3] Arch. f. Kinderh., Bd. XXVII, Hefte 5 u. 6.

**Weight.**—A. Klautsch[1] gives specimen weight-charts of some bottle-fed infants, and, from observations on 200 infants, concludes that dentition is frequently a disturbing factor in the gain in weight of artificially fed infants, without necessarily being a cause of sickness; eruption of one or more teeth was often accompanied by either loss in weight or failure to gain for a short time. Of more decided influence were acute illnesses, especially of the alimentary tract; but in convalescence there was a more rapid gain than before, compensatory in nature. The author's observations of children in the second year of life confirmed Schmid-Monnard's, which showed that, as a rule, the increase in weight from February to May is not so rapid as from June to January.

**Premature Infants.**—E. Deutsch[2] reviews the care of premature infants in lying-in hospitals, and gives the indications for the use of the incubator, describing the different varieties. N. Berend[3] gives extensive consideration to the care of new-born infants in lying-in hospitals, laying down a few general directions with regard to medical observation by a pediatrist, education of the mothers by printed slips, proper clothing of the infants, the care of the navel, and the avoidance of washing the mouth. J. D. Voorhees[4] treats of the care of premature babies in incubators, mentioning as the 4 problems the maintenance of a proper temperature, the prevention of exhaustion, the administration of the proper amount and kind of nourishment, and the avoidance of infection. The details of each of these are given, with the statistics of 336 premature infants at the Sloane Maternity, New York: 106 were put in the incubator, and 29 died within 4 days of birth; of the remaining 77, who survived the fourth day, 14 died before leaving the incubator. Very complete tables give the different points connected with each case.

## MILK AND INFANT-FEEDING.

A. Baginsky and P. Sommerfeld[5] detail the production and **control of milk** used at the K. u. K. Friedreich Kinderhospital in Berlin. Dissatisfaction with the supply of milk from ordinary sources led to the establishment in connection with the hospital of a dairy in which only healthy cows are put, and careful supervision of the stable, fodder, cows, and milk is maintained; the milk, as soon as it is drawn, is immediately carried to the milk-room, where it is filtered and cooled to below 10° C.; it is then carried to the hospital and put in charge of an attendant whose sole duties are with reference to it; this attendant, whose hands and clothes are scrupulously clean, makes record of the temperature of the milk, condition of the receptacles, etc., and any irregularity is reported to the one in charge of the stable for correction; the milk to be supplied to infants is prepared in bottles according to the directions for each child and then sterilized. Sommerfeld reports especially on the examination of the milk, physically, chemically, and bacteriologically; by means of an instrument devised by him, determination is made several times a week of the solid impurities, and such are the precautions of cleanliness that rarely is there sufficient impurity to be weighed. [The Hospital is to be congratulated on having an endowment which enables it to take such a step.]

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[1] Arch. f. Kinderh., Bd. XXVII, Hefte 3 u. 4.

[2] Arch. f. Kinderh., Bd. XXVIII, Hefte 3 u. 4.

[3] Arch. f. Kinderh., Bd. XXVIII, Hefte 5 u. 6.

[4] Arch. of Ped., May, 1900.

[5] Arch. de Méd. des Enfants, May, 1900.

E. Wende,[1] in a report on the cause and prevention of **infant mortality,** treats of the control large cities should take of their milk-supply; the paper is full of many valuable suggestions. Public sentiment in this country is gradually awaking to the significance of the figures of infant-mortality, but it will probably be some time before the subject receives the same attention as is now given it in Europe. Its close dependence on the milk-supply is too well known to need much argument in its behalf. A special committee has been appointed in England,[2] where the subject is considered of national importance, to consider general improvements in the supply. In Liverpool, a city with a very bad milk-supply, where the death-rate of infants is 42% of the entire mortality of the city, the municipality is about to establish a depot for the preparation of sterilized milk of the best quality, which will be sold at a low price to bring it within reach of the poor.

G. W. Goler,[3] the Health Officer of Rochester, N. Y., gives a table showing the **death-rate of children** under 5 years in the largest cities of New York State. There has been, in general, in the past 10 years, a steady lowering of the rate, the greatest decrease (13.5%) having occurred in Rochester, where an ordinance has been passed limiting the number of bacteria in the milk sold in the city to 100,000 per cubic centimeter. For 2 years before this, there were stations in the city where the poor could obtain at cost good milk of definite composition. The fall in the death-rate may, in part at least, be justly attributed to the care of the milk-supply.

H. Carter,[4] the Milk Inspector for the city of St. Louis, reported **analyses** of 331 samples of milk, 176 of which, or nearly 53%, were adulterated with water to the extent of from 5% to 45%; aside from the fraud, the great danger is in the liability to spread typhoid fever. Formalin is being used more than boric acid as a preservative. The highest percentage of fat found was 4.8; the lowest, 1.

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[1] Pediatrics, vol. IX, No. 4.

[2] Phila. Med. Jour., April 28, 1900.

[3] Arch. of Ped., Oct., 1899.

[4] St. Louis Courier of Med., vol. XXI, No. 3.

G. L. Eastes [1] contributes a paper on the **pathology of milk,** based on an examination of 188 specimens, with the surprising results that 5.3% of the specimens contained tubercle bacilli, 30% had pus, 48.7% had mucopus, and 75% showed streptococci. The presence of any of these is sufficient to make the milk unfit for food. Other germs that have been found in milk are Staphylococcus aureus, the diphtheria bacillus, and Bacterium coli. Blood may be present normally very early in lactation, but after this it signifies acute inflammation of the mammary gland or ducts; mucus with an excess of leukocytes, the mucopus, indicates inflammation of the ducts. Stall-fed cows are more likely to give contaminated milk than are cows fed in pasture.

J. G. Adami [2] refers to **bovine tuberculosis,** in its spread among cattle, in the possibility of its spread to man, and in the ways by which this transmission may occur. He finds that bovine tuberculosis spreads easily among cattle; that, inasmuch as the bacillus of bovine tuberculosis differs distinctly from the bacillus of human tuberculosis, and as cattle are relatively insusceptible to the bacillus from human beings, a positive opinion can not be given as regards tuberculous cattle infecting human beings; clinical evidence, however, favors this, and the most probable mode of infection is through the milk from tuberculous cows; in such milk, without macroscopic or microscopic lesions of the udder, tubercle bacilli have been found. The importance of stamping out tuberculosis in cattle is thus seen to be great.

Rabinovitch and Kempner [3] had similar experiences in finding tubercle bacilli in milk without discoverable foci of disease, 8 such cows that reacted to tuberculin giving milk that contained the bacilli.

Jemma [4] studied on animals the pathogenic activity of the bacteria found in milk, which exert on the casein a proteolytic action, and are called the casein ferments. V. C. Vaughan and J. T. McClymonds [5] give a summary of their investigations on some **bacteriologic poisons** in milk and milk-products; in 49 samples of American green cheese a bacillus of the colon group was found in every sample. The clinical observation that opium does harm if given in the vomiting and purging from tyrotoxicon poisoning led to experiments on guinea-pigs with the bacillus obtained from cheese; 0.5 cc. of a beef-tea culture killed only those animals that had received ¼ gr. of morphin hypodermically.

A. G. Young [6] states that **formaldehyd** tends to impair the nutritive value of milk, interferes with digestion, and under some conditions might cause dangerous and even fatal results, and its use should therefore be forbidden by law as a preservative for milk. H. E. Annett [7] comes to similar conclusions, after having fed some kittens with milk preserved with boric acid, and others with milk preserved with formaldehyd; disturbances of nutrition, and in some cases death, resulted.

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[1] Brit. Med. Jour., Nov. 11, 1899.

[2] Phila. Med. Jour., Dec. 30, 1899.

[3] Zeit. f. Hyg. und Infectionskrankh., Bd. XXXI, Heft 1.

[4] Rev. mens. des Mal. de l'Enfance, vol. XVIII, No. 1.

[5] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.

[6] Sanitarian, vol. XLVIII, No. 361.

[7] Lancet, Nov. 11, 1899.

J. M. Whitfield[1] gives tests for the detection in milk of the following preservatives: formaldehyd, boric acid and borax, sodium carbonate, salicylic acid.

C. Baron,[2] writing on the **dirt-content of market milk,** points out that a milk diluted with pure water, aside from the fraud to the purchaser, is less injurious to the health than an undiluted milk rich in dirt and bacteria. Renk's method for the detection of filth is described, according to which the author has found the milk in Dresden to contain an average of 6 mg. of dry (= 30 mg. of fresh) filth per liter. On this basis it is estimated that there are consumed annually in that city 2200 pounds of cow-excrement. The effect of filtration is to hold back practically all solid impurities and many of the bacteria, while it lowers the fat so slightly that this disadvantage may be disregarded. The different materials used in the construction of filters are enumerated, and one made of gravel is most highly recommended. The value of cleanliness in milking and in handling milk is emphasized, and some of the regulations governing the sale of nursery milk in Frankfort and Berlin are given. The effect of these in improving the milk-supply is shown by the fact that in 1896 examination of 730 samples of milk resulted in the condemnation of 28%, while in 1898 there were only 3% rejected in 1500 samples.

R. G. Freeman[3] found that the **bacteria** of milk to a very large extent rise with the cream; one sample that had contained 7750 bacteria per cubic centimeter showed, after the cream had risen, only 20 per cubic centimeter. The practical application of this, although limited, is easily seen to be of great importance in feeding difficult cases, when milk that has been heated is not well digested; the cream could be separated and sterilized and then mixed with the milk.

On the subject of **infant-feeding** some very good editorials appear in the Archives of Pediatrics, Nov., 1899. The general practitioner is too prone to believe that "there are many children who can not take milk," the mistake usually made in such cases being that too strong mixtures are given, the secret of successful feeding being to give a food which the child can digest. Rules for such feeding can only be very general, as each child must be studied by itself. In order to know what the child is getting, the percentage method furnishes the most accurate means; formulas have been put forth[4] by which these percentages may be calculated at home, where the preparation of the milk is sometimes desirable. As stated in another editorial in the same journal for Feb., 1900, some infants fail to thrive on percentage mixtures from the laboratory, while a home modification aiming at the same percentages may be suitable. This latter point is set forth by L. Starr,[5] who gives the results of a clinical study of laboratory milk in substitute infant-feeding. He enumerates the advantages of laboratory milk, among which are the care with which the cows are selected and the cleanliness enforced in collecting and handling the milk, thus reducing the number of bacteria to a minimum; with laboratory milk it is also possible to give an infant a milk identical in composition with human milk, and to vary the percentage at will. In spite of the latter theoretic advantages, the author's experiences in 64 cases are shown by classifying them in 3 groups—the satisfactory, the partly satisfactory, and the unsatisfactory. Of the satisfactory, there were 3 cases; the partly satisfactory, numbering 16, showed no definite disease symptoms, but a failure to gain properly, with anorexia and other evidences of poor nutrition; there were 35 unsatisfactory cases, in which it was necessary, on account of acute dietetic disorders, to abandon laboratory milk. In the last class, home modification, with, in some instances, percentages approximately similar to the laboratory mixture, was followed by prompt improvement. The author seeks an explanation of this in the laboratory methods of using separated cream to make the mixtures, the centrifuge destroying the natural emulsion of the milk and in some way lessening the digestibility of the proteids, so that a badly nourished child of 10 months, unable to digest a laboratory mixture no stronger in proteids than 1.5%, will thrive on a home mixture containing nearly 3%.

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[1] Med. Reg., Sept., 1899.

[2] Arch. f. Kinderh., Bd. XXVII, Hefte 1 u. 2.

[3] Arch. of Ped., Aug., 1899.

[4] YEAR-BOOK, 1899.

[5] Arch. of Ped., Jan., 1900.

**Gruels** as diluents of cow's milk have long been used to produce by mechanical interference small flocculent curds. This action having been disputed, H. D. Chapin[1] conducted some experiments on the digestion of cow's milk, (*a*) plain, (*b*) diluted with an equal amount of water, and (*c*) diluted with an equal amount of barley-water. The experiments were conducted in test-tubes, and in a dog with an artificial gastric fistula, and showed that not only was the presence of barley-water associated with the presence of smaller curds, but also the curds themselves were more easily digested afterward. The objections to giving cereals to very young infants may be overcome by dextrinizing the gruel; this may be done by using any one of the diastase preparations on the market, or by preparing a decoction of diastase by soaking a tablespoonful of malted barley grains in an ounce of water overnight and straining; 1 tablespoonful of the water will dextrinize a pint of gruel. In the discussion on the paper before the American Pediatric Society, the general trend of opinion was in favor of the use of gruels as diluents. Rotch stated that with percentage feeding such use would not often be necessary. Chapin[2] also describes a tin dipper which he has devised for removing the top milk from quart bottles, the aim being to render more easy and exact the process of home modification.

J. Adriance,[3] in discussing the **chemical composition** of cow's milk, said that instead of the percentage of proteid being 4, as is generally believed and acted upon, a number of investigations showed that the average was not greater than 3.3; but the better the cows are cared for, the higher will be this percentage. In modifying milk for infant feeding it is necessary to remember that as the fat in milk increases, the percentages of the other ingredients diminish materially. The error arising from calculating the proteid at 4% would be very slight when the dilution of the milk or cream did not exceed 1%; but if the dilution amounted to 2%, the result would be nearly 0.25% lower than estimated—an important matter considering what a difference in digestion might result from a variation of only 0.2% in the proteids. In the discussion, J. E. Winters said that many of the failures with infant feeding arose from using too small a percentage of proteid. L. E. Holt expressed the same opinion, giving, as one reason, that too high proteid is used at first, with the result that the digestion becomes seriously impaired, and it is then impossible to make use subsequently of a sufficiently high percentage of proteid. His own method was to start with 0.25% or 0.5% of proteid for the first few days, and then rapidly increase, so that at the end of six weeks the child might be able to take and digest perfectly 1.5% of proteid. V. Adriance also referred to the percentage of proteid as being the key-note to the whole subject of infant feeding.

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[1] Arch. of Ped., Dec., 1899.

[2] Arch. of Ped., Jan., 1900.

[3] Arch. of Ped., May, 1900.

T. M. Rotch [1] mentions some important aspects connected with the **scientific feeding of infants,** and points out that it is necessary for the physician not only to know what modifications of milk to prescribe, but also to know what the child actually gets, otherwise it will be impossible to tell, when the child is doing well, what is the cause of the improvement or the cause when the food disagrees, and then the proper change can not be intelligently made. He protests against the use of unknown milk or cream as being likely to be unsafe from a bacteriologic standpoint, and as furnishing unknown quantities of fat and proteid for the home modifications. Maternal anxiety to be sure that the child will get a sufficiently strong food often leads to the addition of a little more milk or cream, with the result that the percentages are changed greatly.

Biedert [2] gives an extensive monograph on the dietetic treatment of the digestive disturbances of children.

H. von Ranke, [3] refers to several cases of undersized new-born infants fed successfully for the first 2 months of life on asses' milk, in spite of the low fat-content which all analyses show; this lowness in fat renders it unsuitable for use after the second or third month. The reason why new-born infants digest it so much better than diluted cow's milk is in the relation between the casein and the albumin, as shown by Koenig's table, in which the casein of cow's milk is to the albumin as 100 : 17.5, while in woman's milk it is 100 : 122, and in asses' milk 100 : 231. The great obstacle to a more extended use of **asses' milk as a food for infants** during the first 2 months of life is its cost, as one animal gives about enough for one child, and the period of lactation is not a long one.

L. Netter [4] gave test-meals (100 gm.) of **sterilized milk** to 3 infants between the ages of 6 months and 1 year who had been fed on sterilized milk from birth. Half an hour after the beginning of the feedings the contents of the stomach were withdrawn and analyzed, and the author arrives at the conclusion that the chemistry of such cases does not differ from that of breast-fed infants; that sterilized milk is as well digested as mother's milk; that it excites the same degree of chlorin secretion; that it does not form a hard curd and does not produce a greater degree of fermentation.

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[1] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.
[2] Arch. f. Kinderh., Bd. XXVII. Hefte 3 u. 4.
[3] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.
[4] Progres méd., vol. x, p. 225.

W. Camerer[1] made extensive studies on digestion in infants and its influence on metabolism.

L. Ballin[2] studied in Heubner's clinic the activity of **gastric digestion** in dyspeptic infants, with special reference to the relation between the duration of digestion—that is, the length of time it takes the stomach to empty itself—and the degree of the dilution of the milk. Equal volumes of different dilutions were given, and it was found that the more concentrated required a longer time, but not in direct proportion to the degree of dilution, the proportion being less. The increase in time required seemed dependent on the proteid-content rather than on the fat; of unequal quantities, the larger bulk was relatively more quickly digested than the smaller.

B. K. Rachford[3] experimented on the **pancreatic digestion of casein,** using the pancreatic juice and bile of rabbits. Tables show the results of the experiments, which, briefly stated, are that casein proteolysis by the pancreatic juice is assisted to a slight extent by the presence of a solution of maltose or of milk-sugar, to a considerable extent by the presence of lime-water or sodium carbonate, and that combined hydrochloric acid, if bile is present, also favors the digestion.

H. Cramer,[4] after investigating the **nutrition** of the new-born during the 10-day period of loss of weight, found that in artificially fed infants the loss was greater and the subsequent recovery slower than in breast-fed infants during the same time; the optimum mixture for the bottle-fed seemed to be milk 1 part, water 2 parts, milk-sugar 4%, which, as Finkelstein remarks, exceeds the natural food in caloric value. Investigation of the negative pressure during the act of sucking showed that at the breast a child must develop a negative pressure of from 13 cm. to 69 cm. of water, according as the breast is an "easy" or a "slow" milker, while from the bottle, milk will flow with a pressure of from 3 cm. to 8 cm. only. The author points out that a breast-fed child will therefore be more tired after nursing, and will sleep better than a bottle-fed child, with probably consequent influence on metabolism.

O. Heubner[5] refers to the careful and extensive researches that are making the **feeding of infants** scientifically exact, a standard being furnished by nature in a healthy breast-fed infant. For healthy older children there is no such standard, and only the highest praise can be given to such work as Camerer has done in studying most thoroughly the metabolism of his 5 children over a long period of time. After referring to other investigators who have studied the needs of the growing child for the different food-stuffs, Heubner concludes with the remark that a healthy child should eat "rather somewhat lightly than too abundantly."

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[1] Jahrb. f. Kinderh., Bd. LI, Heft 1.

[2] Ibid., Heft 6.

[3] Arch. of Ped., June, 1900.

[4] Deut. med. Woch., No. 2, 1900; Jahrb. f. Kinderh., Bd. LI, S. 689.

[5] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.

A. Czerny [1] gives an interesting report of the effects, in his experience, of a strengthening or **roborant diet,** meaning by that the feeding to children of large amounts of meat, milk, and eggs, to the exclusion of vegetables. He has seen obstinate constipation and anemia often depend on the too-long-continued use of milk for children in the later part of infancy, in such amounts as to interfere with the taking of a more varied dietary. Anorexia and an obstinate diarrhea, which is prone to recur, are sometimes caused by the strengthening diet; and when meat has been the main article of food, the urine is usually high-colored, precipitating urates and uric acid, and often causing in little girls a vulvitis. The author mentions a hitherto undescribed type, the result of this diet, seen in children of well-to-do parents, there developing a yellowness of the skin, associated with more or less adiposity, jaundice being easily excluded. Skin eruptions and disturbed sleep sometimes follow the diet also. The author doubts the roborant diet's value in scrofula, and states that while it is impossible to say that the diet predisposes to the infectious diseases, yet many of the latter often directly depend on the state of nutrition, and that a proper diet, rather than one with an excess of proteid, would be more likely to act as a preventive. Overfeeding would seem to be favorable for the development of scarlet fever in older children, as all the malignant cases ending fatally in 24 to 48 hours seen by Czerny were in such children. [The author's points are well taken, but he does not mention the difficulty sometimes experienced in overcoming the reluctance of such children to take with relish a more general diet.]

## INFECTIOUS DISEASES.

**Diphtheria.**—W. Bloch and P. Sommerfeld, [2] in studies on the pathogenicity of the Loeffler bacillus, have verified the accepted statements with reference to the germ, their article being a good exposition of the present status of the bacteriology of diphtheria. The main points have all appeared in the pages of the YEAR-BOOK in previous years, but it is of interest to mention a few upon which emphasis is laid. From a study of 436 cases the authors state that the Loeffler bacillus was never found in pure culture, but always associated with other bacteria, among which streptococci played the greatest part. The two doctrines concerning the relation of streptococci to septic diphtheria are given, the one being that the streptococci increase the virulence of the diphtheria bacilli and cause sepsis by gaining access to the circulation; the other is that the diphtheria toxin, by its effect on the organism, prepares the way for an invasion by streptococci. The authors think that the latter view agrees more nearly with clinical experience, for even in the mildest cases streptococci are present with the bacilli, and general infection does not always follow; and, further, in cases presenting the beginnings of severe sepsis, large doses of antitoxin frequently save life. They therefore state that the diagnosis of septic diphtheria can not be based on the results of cultures from the pharynx. From cultures of the heart-blood made an hour after death in 11 cases of septic diphtheria, and from autopsies on the same, they assert that the realm of septic diphtheria clinically does not coincide with that in pathology in which the diagnosis is based on the presence of streptococci postmortem in the blood. With regard to the distribution of the bacilli in the internal organs, 20 autopsies gave 4 positive results from the heart-blood, all the cases giving positive cultures from the lungs; in 14 of the cases the spleen was positive 5 times, the kidneys 3 times, and the liver once.

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[1] Jahrb. f. Kinderh., Bd. LI, Heft 1.

[2] Arch. f. Kinderh., Bd. XXVIII, Hefte 1 u. 2.

H. Braun and G. Thiry [1] discuss under the term **septicemic diphtheria** the generalization of the diphtheria bacillus in the body, stating that it has never been demonstrated in the blood of human diphtheria patients. It has been shown by many investigators that the injection of a pure culture of the bacilli into animals is rarely followed by their presence in the blood or internal organs, but that this often happens when other germs are injected at the same time and place; certain chemical substances, as atropin and pilocarpin, seem to have a similar influence. Cultures from the organs of diphtheria autopsies in human beings rarely give the bacillus in pure culture, the germs most commonly associated being streptococci and staphylococci, these being more common in the bronchopneumonias than are the pneumococci or pneumobacilli. According to Martin, the primary seat of diphtheria in many cases is the lung, the trachea and larynx being involved secondarily; the authors observed a case in which the pharynx and larynx were only slightly affected in proportion to the degree of toxicity shown, and they infer that the pseudomembrane is not the only place for the formation of the toxin, but that the bacilli penetrate, in such cases, into the blood, causing a diphtheric septicemia, and elaborate their toxin there. On this point L. Tollemer [2] states that the diphtheria bacilli are distributed in the body more widely than is generally thought; in 49 autopsies after diphtheria he obtained cultures 12 times from the heart-blood, 18 from the heart muscle, 19 from the spinal cord, 3 from the cerebrum, 31 from the lungs, once from the liver, and 7 from the spleen, while cultures from the spinal fluid and kidney were always negative. Inoculations of these cultures, with and without antitoxin, showed the bacilli to be true ones, and virulent. The severity of the cases and the pathologic findings did not explain why the dissemination occurred in some and not in all cases; that it was not agonal is shown by the irregularity of the distribution. It is thought that the presence of the bacilli in the heart and nervous system may determine the development of paralysis through the production of the toxin in these locations, and the administration of antitoxin is strongly urged.

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[1] Gaz. des Hôp., 1899; Jahrb. f. Kinderh., Bd. LI

[2] Gaz. des Mal. inf., No. 18, 1899.

Edgeworth [1] reports a case of diphtheria terminating fatally from a **clot in the basilar artery,** the exciting cause of the formation being probably a choking and coughing spell during eating. [It is not stated whether cultures were taken from the thrombus, an examination which would have been of great interest.]

Charrin and Levaditi [2] have shown that **pancreatic substance** exerts a destructive action on the power of the diphtheria toxin. [Without detailing the experiments, it may be said that they explain, in part at least, why the large amount of bacilli-laden saliva which is swallowed in almost every case of diphtheria does not lead to an autoinfection; for if the bacilli themselves are not destroyed, they are rendered practically harmless by the neutralization of their toxin.]

T. G. Brodie, [3] after experiments on cats with the diphtheria toxin, concludes that the primary cause of death within 48 hours after an injection of the toxin is **paralysis of the blood-vessels,** with the consequent fall in pressure; whether this is due to direct action on the muscular coat or on the central nervous system is not made out. J. Biernacki [4] explains, from the clinical standpoint, the symptoms of diphtheria in the same way.

J. W. H. Eyre [5] describes an epidemic of diphtheria disseminated by **milk,** the patients being pupils in a large school; diphtheria bacilli were obtained by cultivation from the cream and centrifuged milk; inoculation experiments showed the bacilli to be virulent. The source of contamination of the milk could not be ascertained.

T. Hubbard [6] refers to the association of **peritonsillar abscess** with diphtheria, and reports several cases; mild diphtheria may sometimes be marked by a severe attack of quinsy, and in these mixed infections it is probable that the virulence of the different bacteria, with the age and natural resisting power of the patient, determine which type of infection shall predominate. Incision should be made through the softened tissue, the vascular tissue being avoided as much as possible.

F. Villy [7] discusses **vomiting and cardiac failure** in connection with diphtheria, tabulating 177 cases observed with reference to the onset of cardiac irregularity, which occurred in 87.5%, the average onset being on the eighteenth day; 1000 cases were analyzed with reference to the presence and onset of paralysis, which affected 16.2%, the average day of onset being the twenty-eighth; 50 cases of vomiting followed by cardiac failure and death had an average day of onset of the eighth. A number of case histories are given. From the clinical evidence the following deductions are made: (1) Signs of heart failure are much more common than are paralyses in other parts, and have an earlier date of onset; (2) they are such as may be ascribed to muscular failure; (3) when vomiting occurs, heart failure generally follows; (4) the date of onset of vomiting and heart failure is distinctly anterior to that of paralysis in other parts. From the pathologic data the conclusions are: Evidence of degeneration and inflammation of the mucous membrane of the stomach is constantly present, and is often accompanied by hemorrhages. The heart muscle is constantly found to be in a degenerated condition, and hemorrhages are commonly present. The combined evidence points to the vomiting and cardiac failure as being due to the local changes in the heart and stomach, and not being dependent on nerve lesions. Vomiting, by the strain thrown on the heart, and perhaps reflexly, may produce heart failure.

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[1] Lancet, No. 3954; Jahrb. f. Kinderh., Bd. LI.

[2] Gaz. des Hôp., No. 40, 1899; Jahrb. f. Kinderh., Bd. LI.

[3] Brit. Med. Jour., Nov. 4, 1899.

[4] Ibid., Dec. 30, 1899.

[5] Brit. Med. Jour., Sept. 2, 1899.

[6] N. Y. Med. Jour., vol. LXX, No. 16; Arch. of Ped., Feb., 1900.

[7] Med. Chron., third series, vol. I, No. 6; Arch. of Ped., Mar., 1900.

F. V. Szontagh [1] reports a case of diphtheria in which the use of **antitoxic serum** was followed by a severe attack of **fever,** swollen joints, measles-like eruption, and intense general pains, recovery following after 2 months; the author attributes the sequel to a personal idiosyncrasy, and states that while in this case the remedy was worse than the disease, it has not weakened his faith in the antitoxin.

H. Kraus [2] reports a case of diphtheria in a girl of 9 years, **paralysis** developing on the fourteenth day. There was also a curious edema, affecting the thighs, face, and trunk, but especially the arms, which were swollen like cones with the bases at the elbows; the nerve-trunks were tender, and the cause of the edema was held to be the polyneuritis. Recovery ensued.

A. Schuetze [3] reports a case of diphtheria in a woman, in the course of which on the tenth day there appeared an **erythema nodosum,** with joint-swelling, the antitoxin not having been administered; the author reports the case to show that all the complications after antitoxin injections ought not to be ascribed to the serum.

W. H. Park [4] has endeavored to separate the **diphtheria antitoxin from the blood-serum,** in order to avoid the occasional sequels of injection, such as rashes, fever, and joint-pains. Using as a basis the fact that as the blood of the horse increases in content of antitoxic units the globulin also increases, and seems to be closely connected with the antitoxin, the "antitoxic globulin" was separated and used in 48 cases, with the same results that attend the use of the antitoxic serum as regards recovery and rashes, but with the disadvantage that there was in one-fourth of the cases a somewhat greater local reaction following the injection. The dried globulin, which is easily soluble in water, has retained its antitoxic properties for 2 months, and probably will not deteriorate so quickly as the serum.

J. L. Duenas [5] gives the experiences of himself and other Cuban physicians in the use of **antitoxin,** all being advocates of the treatment. O. Wenner [1] gives the results of the treatment of diphtheria with the antitoxin in the Children's Hospital at Zuerich, comparing his statistics with those collected by Baer and Paperna in the same service for the pre-antitoxin period. We commend his article especially to those who honestly doubt the efficacy of the antitoxin, hoping that it will lead them to use the antitoxin as Wenner has used it, for they will then become convinced of its great value. The article is a dispassionate statement of fact, of which a good summing-up is the following table:

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[1] Arch. f. Kinderh., Bd. XXVIII, Hefte 5 u. 6.

[2] Jahrb. f. Kinderh., Bd. L.

[3] Deut. med. Woch., 1899, No. 49; Jahrb. f. Kinderh., Bd. LI, Heft 6.

[4] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.

[5] Arch. of Ped., Dec., 1899.

| | PRE-ANTITOXIN. | | ANTITOXIN. |
|---|---|---|---|
| | *Baer.*<br>1874–1891. | *Paperna.*<br>1891–1894. | *Wenner.*<br>1894–1898. |
| Total number of cases | 690 | 149 | 432 |
| Deaths | 302 | 60 | 44 |
| Mortality rate | 43.8% | 40.3% | 10.18% |

Mayer [2] states that when there is a tendency toward involvement of the larynx in the diphtheria seen in Fuerth, where septic cases are rare, one injection of antitoxin is usually sufficient to check the spread of the process. A. J. Turner [3] says that the only objection to giving an unnecessarily large dose of antitoxin is its cost, as there need be no fear of harm in giving an overdose.

W. H. Park [4] has found that for 15 years before the use of antitoxin the average number of deaths yearly was 2373 in New York; for the 4 years since the general use of the antitoxin the annual average has been 1341. He recommends in mild cases seen early, 1000 units; in mild cases seen late, 1000 to 2000 units; in severe cases seen early, from 2000 to 4000 units; and in severe cases seen late, an initial dose of not less than 3000 to 4000 units.

Wright [5] reports 258 cases with a mortality of 22%. He ascribes this high percentage to the fact that many were seen in consultation after the disease had made considerable headway, the mortality in cases injected in the first 48 hours being 11.8%, while of those seen later, 72% died. He never uses less than 1500 units and rarely more than 3000 units at one dose, being guided solely by the physical condition, extent, and location of the membrane.

B. R. Shurley [6] reports 100 intubations, with antitoxin and a mortality of 31. A. Anderson [7] treated 258 cases of diphtheria with antitoxin, 41 dying, or 15.8%.

E. Rosenthal [8] shows that the position of the profession on the use of antitoxin is due to the experience of pediatrists; he addressed communications to over 4000 physicians, and found them practically unanimous in favor of it; statistics from health boards and hospitals in 157 cities gave a mortality of 38% without and 14% with antitoxin.

Villy [9] recommends large doses of antitoxin, sufficient to produce a reaction in 12 hours, and says that an initial dose of from 8000 to 12,000 units in severe cases, even when seen late, will often give surprisingly favorable results, when the ordinary dose of 2000 to 4000 would have scarcely any effect. At nearly the other extreme is the plan adopted by J. H. Musser,[1] who has treated a short series of cases with complete success, giving to children under 8 years 500 units, repeated every 6 hours if improvement does not follow.

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[1] Arch. f. Kinderh., Bd. XXVII, Hefte 1 u. 2.

[2] Münch. med. Woch., Nov. 21, 1899.

[3] Brit. Med. Jour., Dec. 30, 1899.

[4] Phila. Med. Jour., Mar. 31, 1900.

[5] Pediatrics, Mar. 15, 1900.

[6] Jour. Am. Med. Assoc., May 19, 1900.

[7] Quart. Med. Jour., Feb., 1900.

[8] Phila. Med. Jour., June 9, 1900.

[9] Med. Chron., Jan., 1900; Phila. Med. Jour., April 28, 1900.

**Measles.**—Jurgenssen [2] asserts that very **young infants are immune** to measles, and states that of 41 exposed to the disease, all over 6 months of age contracted it, while the 25 under 5 months were not affected.

A. Bartch [3] reports an instance of **intra-uterine infection** in an infant born at term when the mother had a rash of measles; in 3 days the infant, who had coryza and cough from birth, developed a measles rash with fever, recovering in 6 days.

V. Adriance [4] gives the statistics of an **epidemic** of measles, comprising 96 cases. The seriousness of the disease in a children's hospital and the impossibility of checking the spread when it has once gained a foothold were illustrated by the epidemic. Second attacks were seen in 16 of the 96 cases; diphtheria was a complication in 36 cases, 4 ending fatally; the total mortality was 15, or about 16 %; Koplik's spots were observed in 76 %, and in 20 % they were visible before the rash appeared, but the extreme contagiousness of the disease in the catarrhal stage had prevented this foreknowledge from being of value in checking the spread. J. Sobel gave as his belief that the presence of Koplik's spots was as pathognomonic of measles as was the presence of the malarial plasmodium in the blood of malaria, and referred to the value of the spots in differentiating measles from German measles. J. Howland observed the spots in 84 % of an epidemic of 61 cases of measles. La Fetra found them to be of value in distinguishing the disease from antitoxin rashes. R. G. Freeman had observed the spots in about the percentage mentioned, but he had also seen them in children who had not developed the rash subsequently, and he therefore thought it not safe to base a positive diagnosis on their presence and then expose a child on that account to infection with measles.

Rolly [5] reports the concurrence of **measles and scarlet fever** in an infant 1½ years old, the measles rash following that of scarlatina by 3 days. Both diseases were well marked, and apparently had no modifying effect on each other. The patient recovered. Usually the prognosis is better when there is a longer interval, if the scarlatina follows the measles, than when the reverse occurs.

F. R. England [6] reports a case of measles followed, as the rash was fading, by **meningitis,** resulting fatally in 38 hours. Cultures from pseudomembrane in the nose gave pure growth of a diplococcus having the characteristics of the meningococcus. Cultures from other patients with measles in the same house gave the same germ with staphylococci. Scarlet fever in the house suggested a possibility of that, but the symptoms did not support that diagnosis.

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[1] Univ. Med. Mag., Mar., 1900.

[2] Deut. Arch. f. klin. Med.; Arch. of Ped., Sept., 1899.

[3] Arch. of Ped., July, 1899.

[4] Arch. of Ped., Feb., 1900.

[5] Jahrb. f. Kinderh., vol. L; Arch. of Ped., Mar., 1900.

[6] Montreal Med. Jour., vol. XXVIII, No. 11; Arch. of Ped., Mar., 1900.

Sotow[1] reports 3 rare **complications** following measles: a child of 12 months presented general tremors for 6 weeks, recovering; a boy 4 years old recovered after presenting mania followed by epileptiform attacks and then periods of apathy alternating with agitation; a girl of 9 years complained of failing vision, and became almost completely blind, the ophthalmoscope showing miliary hemorrhages in each retina.

J. H. Adams[2] reports an attack of measles in a 10-days-old child, the other children in the family having had measles 13 months earlier. Investigation by the physician failed to disclose another case of measles in the small town in which the family lived, and then it was found that the infant was wrapped immediately after birth in a **blanket** which had been used by the children during their attacks of measles, after which it had been steamed and put away for **13 months.** The author ascribes the attack to a persistence in the blanket of the infective principle of measles.

M. Cohn[3] points out that **Koplik's spots** are to be distinguished from the red patches at the beginning of measles, the latter being a true eruption of the disease, while the former, bluish-white, are minute points of desquamation; the author detected them in 16 out of 22 cases,—a somewhat smaller proportion than Slawyk, Havas, and others,—while Knoespel found them in all of 41 cases. Cohn thinks that while their absence does not exclude measles, their presence is pathognomonic.

**Rötheln.**—A. Tobeitz[4] gives a brief report of the latest **epidemic** of rubeola (rötheln) in Graz. The epidemic lasted from December, 1898, until July, 1899, with a total of 719 cases, and it could be compared with a measles epidemic comprising 3273 cases and running from June, 1898, to June, 1899. As compared with rötheln, the greater infectivity of measles is thus shown; measles also attacked infants oftener than rötheln, while the latter showed a larger percentage of adults, probably because they had not been protected by previous attacks in childhood. Second attacks were seen in 2.5% of the total number of rötheln cases, surpassing that of the measles epidemic, in which only 0.46% had a return of the disease. The author observed 27 cases in which the incubation period could be definitely fixed, and he found this to range from 4 to 25 days, so he concludes that personal and other influences are so powerful that there is no regular period of incubation. Enanthem on the palate was not seen, but the cervical glands, both anterior and posterior, were always enlarged; complications and scaly desquamation were absent.

F. Theodor[5] discusses the **specificity** of rötheln, second attacks of the same, and their relations to measles and scarlatina. He reports an epidemic in Königsberg so extensive that even the most skeptical had to admit the identity of the disease; the epidemic was characterized by a number of relapses, by a tendency to attack adults, especially women, and by mixed infections with scarlatina and measles, of which the author details instances.

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[1] Jahrb. f. Kinderh., Bd. L, 1899.

[2] Arch. of Ped., July, 1899.

[3] Therap. Monatsch., 1899, Heft 11; Jahrb. f. Kinderh., Bd. LI, Heft 6.

[4] Arch. f. Kinderh., Bd. XXVIII, Hefte 5 u. 6.

[5] Arch. f. Kinderh., Bd. XXVII, Hefte 1 u. 2.

**Scarlet Fever.**—J. W. Stickler[1] took **mucus** from the throats and buccal cavities of scarlatinal patients, and, after mixing it with carbolic acid 1 : 600, **injected** it **subcutaneously** into 10 children, causing scarlet fever in each child; the average period of incubation was 32 hours and 13 minutes, the shortest being 12, and the longest 72, hours; the author concludes that this proves that the mucus of the throat and mouth contains the germ of scarlet fever, and that disinfection should be along this line with great care. Finding that he was producing scarlet fever, the author terminated his experiments. [But it is difficult to see what impelled him to start them.] All the cases recovered.

R. H. B. Gradwohl[2] examined 7 cases of scarlet fever with reference to the presence of the **diplococcus** described by W. J. Class as being the cause of the disease, and found it in all the cases; the author is convinced of its specificity, and gives an extended description of the germ.

J. P. C. Griffith[3] reports 4 cases of **miliary scarlet fever,** showing that a miliary eruption may be present in a mild case, with a slight rash and without extensive peeling. The development of miliaria probably depends on some peculiarity of the skin of the patient:

W. J. Class[4] reports on his experiments to obtain an **antitoxin** for Diplococcus scarlatinæ, in which he was successful, using swine as the largest animals susceptible; blood-serum obtained from a female inoculated with the germ protected guinea-pigs from cultures of the same germ, the control animals dying in 6 or 7 days.

A. Steffen[5] contributes an interesting article on scarlet fever, summing up, from his experience in over 1200 cases, the present status of knowledge on the subject, illustrating some of the points by reference to certain cases. One of the most interesting points is the observation that the germ spreads less easily between bodies whose cells and tissues are of similar characteristics, than between those who are of different families, the poison in the latter instance having attained to a different degree of intensity.

J. F. Schamberg[6] tabulates the examinations of 100 cases of scarlet fever with reference to the **enlargement of the lymphatic glands,** finding enlargement of the inguinal glands in 100%, of the axillary in 96%, of the maxillary in 95%, of the posterior cervical in 77%, of the anterior cervical in 44%, of the submaxillary in 36%, of the epitrochlear in 26%, and of the sublingual in 25%. The enlargement did not seem to depend in degree on the severity of the rash, but rather on the intensity of the toxemia. A control examination of 25 cases of diphtheria showed that there may be a general enlargement of the glands in that disease, so that the study does not help much in differential diagnosis from this disease, its main value lying in recognizing scarlet fever from scarlatiniform eruptions due to various toxins or drugs, in which the glands are not so universally enlarged.

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[1] Med. Rec., Sept. 9, 1899.

[2] Phila. Med. Jour., Mar. 24, 1900.

[3] Phila. Med. Jour., May 12, 1900; also Jacobi's Festschrift.

[4] Phila. Med. Jour., June 23, 1900.

[5] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.

[6] Ann. of Gyn. and Ped., vol. XIII, No. 3; Arch. of Ped., April, 1900.

A. J. Rosenberry [1] reports a **relapse in the third week** of scarlatina.

W. G. Nash [2] observed 3 cases of scarlet fever without eruption occurring in an epidemic of 9 cases, all being traced to a common **milk-supply**; the symptoms in the 3 cases were sore throat, fever, and desquamation. H. W. Irvine [3] reports a similar case in a girl of 18 years.

Although scarlatina is rare in **early infancy**, Josias [4] reports cases in an infant 6 weeks old and in one a year old.

H. O. Hall [5] furnishes food for thought in his paper calling attention to the fact that scarlet fever does not occur epidemically in countries in which **cow's milk** is not a staple article of food; such countries are Japan and China. In India, where cow's milk is used, and scarlet fever is rare, the custom of nursing the child until it is 3 or 4 years of age is probably the reason. An epidemic of scarlet fever in London was traced to a dairy in which the cows were found to have a vesicular disease of the teats and udders; the pus from these vesicles was injected into healthy calves, who developed similar vesicles, with high fever, diarrhea, and running at the nose. Another epidemic, at Wimbledon, was also traced to a certain dairy. [The paper does not state whether the cows were sick or not; many epidemics have been so traced, but it has usually been found that the milk became contaminated, not from the cows, but from a case of scarlatina in one of the human beings connected with the dairy.]

Egis [6] reports a case of **multiple neuritis** following scarlet fever in a girl 4 years old; diphtheria 18 months earlier had been followed by paralysis, and in the discussion Muratow referred to the tendency of neuritis to relapse, saying that the case could be looked on as such, the scarlatina being the occasion of it. Egis found only one similar case recorded.

Moizard and Ulmann [7] observed in a girl 4½ years old an attack of **phlebitis** starting on the seventh day of scarlet fever and affecting the right axillary vein; cultures from one of the thrombosed veins on the shoulder gave a pure growth of streptococci. Four other cases are collated from literature, a study of these showing that there is no special time for the development of the phlebitis, which does not seem to bear any relation to the severity of the disease, although there was in most cases a grave general infection.

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[1] Therap. Gaz., vol. XXIII, No. 12; Arch. of Ped, April, 1900.

[2] Brit. Med. Jour., 1899, No. 2033; Arch. of Ped., May, 1900.

[3] Ibid., No. 2036; ibid.

[4] Méd. moderne, 1899, p. 251; Rev. mens. des Mal. de l'Enfance, Oct., 1899.

[5] Med. Rec., vol. LVI, No. 20, 1899; Arch. of Ped., Mar., 1900.

[6] Arch. f. Kinderh., Bd. XXVIII, Hefte 5 u. 6.

[7] Arch. de Méd. des Enfants, vol. II, No. 10; Arch. of Ped., Jan., 1900.

In this connection it is interesting to note the case reported by A. T. Bazin,[1] of a boy 21 months old, seen on the twentieth day of a severe attack of scarlet fever, with the head drawn to the right, but rotated to the left, and with the right arm and leg powerless. Thrombosis of the cortical branches of the left middle cerebral artery was diagnosed for the following reasons: (1) The gradual and progressive onset of the paralysis, the arm being affected 24 hours earlier than the leg; (2) the very low blood pressure which had been present for a number of days; (3) that thrombosis is the most frequent postmortem condition found to be the cause of a hemiplegia occurring in the course of specific fevers in children. On symptomatic treatment recovery was complete.

S. Chabade[2] made **cultures from the throats** of 214 scarlet fever patients, 98 of whom had a catarrhal angina, 33 a follicular, while 83 had false membrane. The first group had only streptococci, and in some cases staphylococci, but not one had diphtheria bacilli; 2 of the second group showed the diphtheria bacilli, one culture being nearly pure; in the third group the bacilli were found 11 times, 3 being pure cultures, the remainder having streptococci in association. While the diphtheria bacillus is sometimes found accidentally in healthy throats, without pathogenic activity, the author does not believe that these cases can be so regarded, for of the 103 cases in the last 2 groups not showing the bacilli, the mortality was 38%, while of the 13 cases in which the bacilli were present, the mortality was 62%. In 24 cases of tardy or secondary angina, coming on from 12 to 41 days after the start of the scarlatina, follicular in 6 and pseudomembranous in 18, the Loeffler bacillus was found in 2 of the first and in 16 of the second group, showing that the great majority of these secondary anginas are diphtheric.

H. Kraus[3] refers to the rarity of **perichondritis of the larynx** as a complication of scarlet fever, Rauchfuss having seen it 4 times in 903 cases, and Leichtenstern twice in 467 cases. The author reports a case in a child of 8 years, obstruction of respiration coming on on the sixth day, necessitating intubation and then tracheotomy, the abscess opening into the tracheotomy wound. The case progressed favorably for 10 days, when hemorrhagic nephritis and purulent pleurisy developed, death occurring after an illness lasting a month.

A. Seibert[4] recommends the use of **ichthyol** in scarlet fever, externally in the form of a 10% ointment, and as a nasopharyngeal irrigation in the form of a 5% solution; the ointment is to be thoroughly rubbed into the entire surface of the body every 6 to 12 hours, according to the intensity of the dermatitis. In 56 cases the author has observed the following effects: The swelling of the skin lessens decidedly after the first application; the irritation lessens immediately, and disappears after a few rubbings; rhagades and secondary phlegmonous and erysipelatous skin infiltrations were prevented; in uncomplicated cases the fever falls from 1 to 3 degrees (F.) in a few hours; the restlessness and sleeplessness due to the skin irritation rapidly disappear. After the acute symptoms have subsided one inunction daily may be given through desquamation, which is made less intense and is finished quicker. No toxic symptoms appeared from either the inunctions or the irrigations.

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[1] Montreal Med. Jour., vol. XXVIII, No. 11; Arch. of Ped., Jan., 1900.

[2] Arch. russes de pathol., 1899, p. 208; Rev. mens. des Mal. de l'Enfance, Oct., 1899.

[3] Prag. med. Woch., 1899, No. 29; Rev. mens. des Mal. de l'Enfance, Oct., 1899.

[4] Jahrb. f. Kinderh., Bd. LI, Heft 3.

F. H. Williams[1] found that the period of **desquamation** after scarlet fever was much shortened by the external application of a mixture containing 1 part of glycerin to 7 parts of a 10-volume solution of peroxid of hydrogen, rubbed in once daily to the whole surface of the body except the hairy scalp, a fourth of the body being treated at one time; the disinfectant properties of the mixture may be increased by the addition of HCl enough to make a 0.1% solution.

**Typhoid Fever.**—Moizard[2] reports 3 cases of typhoid fever with rapid onset, simulating **appendicitis,** the symptoms coming on suddenly with high fever, frequent vomiting, and violent pain in the right iliac fossa; the good general condition and absence of marked rigidity of the abdominal muscles were the only points against appendicitis. In one case the appendix was found normal at operation; the continuance of the fever and the appearance of rose spots revised the diagnosis. The Widal reaction is not of help in such cases, not being present so early. The treatment consists in the application of ice to the abdomen, liquid diet, and belladonna and chloroform-water by the mouth, opium being avoided.

G. E. Barksdale[3] reports a case of typhoid fever in a colored boy 4 years old, complicated by **cancrum oris** affecting both cheeks, and terminating fatally.

H. Hawkins and O. Thurston[4] observed typhoid fever in a girl 11 years old, **perforation** occurring on the forty-second day, the laparotomy for which was successful; there followed abscess of the parotid, otitis media of both sides, and swelling of the knee-joint. On the sixty-fifth day, for 11 days, antistreptococcic serum was injected, followed by improvement; but after 24 days, on the one hundred and first day, a relapse of typhoid began, lasting 14 days, after which recovery followed.

A. Moussous[5] reports 105 cases of typhoid fever seen **in hospital service** in the past 10 years with but 1 death; only those cases were included in the statistics in which the diagnosis was undoubted, and many of them were severe. The lines of treatment were: Calomel purges every 3 days up to the tenth or twelfth day; between and after the purges, naphthol or naphthalin, with salicylate of bismuth; quinin daily as the stomach would tolerate it and in proportion to the fever; 2 baths daily with cool water and vinegar; ingestion of as much milk and liquid as possible; and, whenever necessary, bathing. The temperature which the author prefers, as a rule, is about 30° C., but this is modified for each case. The indications for cold baths are stupor and hyperpyrexia.

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[1] Boston M. and S. Jour., Sept. 14, 1899.

[2] Jour. de Clin. et de Therap. Inf., vol. VII, No. 44; Arch. of Ped., Feb., 1900.

[3] Richmond Jour. of Pract., vol. XIV, No. 1; Arch. of Ped., May, 1900.

[4] Lancet, No. 3972, 1899.

[5] Arch. de Méd. des Enfants, May, 1900.

A. Jacobi [1] discusses typhoid fever **in the young,** stating that its comparative infrequence in the first year of life is because the water that infants drink is usually boiled. In children the temperature is not always an indication of the severity of the disease, otitis is not rare, and constipation is present in about half the cases; hemorrhage is seldom seen, and the circulatory system is not so much affected as in the adult; rose spots are absent in 20%; desquamation may be very marked following the fever; aside from the stimulation to their growth, the bones are the seat in about 2% [?] of the cases of either periostitis or osteomyelitis; the nervous system is not often involved. The author quotes his former publications to show that two sets of observations on typhoid fever in children may differ widely in symptoms, course, and complications; with reference to treatment, the author again refers to his choice of warm rather than cold baths, using water of 95° or 90° F.

S. Baruch,[2] the exponent of hydrotherapy in this country, discusses **hydriatic measures** in the management of febrile disorders of infancy and childhood, not limiting the discussion to typhoid fever. He decries the impression which exists widely that the action of water is solely antipyretic, whereas its most important action is to restore tone to the vasomotor and general nervous system. In starting the use of the baths in a case of typhoid he advises having the temperature about 90° F. at first, reducing each successive bath a few degrees until 60° F. is reached. In using hydrotherapy, the great mistake made is the omission of friction.

A. H. Wentworth [3] reports a case of typhoid fever in a girl 4 years old, under observation from the seventh to the twenty-fourth day; the Widal test on the seventeenth and twenty-first days was negative, but the clinical diagnosis was that of a moderately severe typhoid fever. On the twenty-third day symptoms of meningitis appeared, lasting 24 hours, when death occurred. There was no postmortem examination, but a lumbar puncture gave a turbid fluid filled with bacilli having the characteristics of the typhoid bacilli, and cultures of this germ gave agglutination with serum from 2 cases of typhoid in which the Widal test was positive. Shorno [4] and Hugot [5] report somewhat similar cases.

**Acute Rheumatism.**—C. Lachmanski [6] analyzes 73 cases of acute **rheumatic arthritis** and 3 cases of chronic polyarthritis in children. D. J. M. Miller [7] reports a case of acute articular rheumatism in an infant 9 months old, and reviews the literature of the subject, collecting 27 cases, 19 of which he thinks are genuine, the others being doubtful.

Westphal and Wassermann [8] found at the autopsy of a case of acute rheumatism and chorea a recent endocarditis and parenchymatous nephritis. Cultures from the blood, brain, and mitral valve gave a streptococcus which produced **arthritis** in animals, with a tendency to shift from one joint to another; and from the affected joints the same microorganism could be obtained.

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[1] Pediatrics, vol. VIII, No. 12.

[2] Ibid., vol. IX, No. 1.

[3] Arch. of Ped., Nov., 1899.

[4] Arch. f. Kinderh., Bd. XXVIII, Hefte 5 u. 6.

[5] Lyon méd., Jan. 22, 1899; Arch. de Méd. des Enfants, Mar., 1900.

[6] Arch. f. Kinderh., Bd. XXVIII, Hefte 1 u. 2.

[7] Arch. of Ped., Sept., 1899.

[8] Berl. klin. Woch., No. 29, 1899.

**Malaria.**—Moncorvo[1] describes malaria in children as he has seen it in 5000 cases in Rio Janeiro. About 35% of all cases are malaria, showing that children are more susceptible than adults; 4 cases of undoubted intra-uterine transmission of the plasmodium were seen. The colored race is more insusceptible than the white; sex had little or no influence in predisposing. The greatest number of cases occurs in the first 3 months of the year—the summer season. Blood examination was rarely feasible for diagnosis, this being based on clinical features, which are very irregular as compared with those in adults. Among the most prominent symptoms are irregularity of the bowel movements, normal stools alternating in a few hours with thin, slimy ones. The spleen is not always enlarged, but the liver is, and is usually more so than the spleen. Malaria often tends to become pernicious in children. Secondary infections with streptococci and other germs are not infrequent, especially in connection with the respiratory system, and acute nephritis is not rare. Among the obscure symptoms which may be misleading is an erythema nodosum.

B. M. Taylor[2] details a peculiar case of pernicious malarial infection in a boy of 12 years, with wild delirium, subnormal temperature, and slow respirations, the malarial organism being found on the second day; 20 grains of quinin sulphate, dissolved in sulphuric acid, were given hypodermically every 3 hours, and on the next day he was conscious, with normal temperature, pulse, and respiration.

**Epidemic Cerebrospinal Meningitis.**—W. Osler[3] delivered the Cavendish Lecture on the "Etiology and Diagnosis of Cerebrospinal Fever."

R. B. H. Gradwohl[4] observed in an epidemic of cerebrospinal meningitis the case of a pregnant woman who died at the seventh month without having aborted; at the autopsy, in addition to the lesions in the mother's meninges, similar changes were found in the meninges of the fetus, cultures from both giving Diplococcus intracellularis meningitidis, proving it to be clearly a case of intra-uterine meningitis.

L. Zupnik[5] was unable to obtain cultures from the purulent exudate in a case of epidemic cerebrospinal meningitis, while cover-glass preparations showed the presence of diplococci. As the meningococcus grows easily, the author concludes that the germ found was some other diplococcus, and that therefore in this disease the etiology is a varied one, without there being a specific infection. Finkelstein, in reviewing the article, urges [rightly] that the conclusion is based on insufficient grounds, because failure to obtain a growth of the meningococcus has happened to every one who has worked with the germ.

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[1] Pediatrics, vol. VIII, Nos. 3 to 6, 1899.
[2] Med. Rec., vol. LVI, No. 23; Arch. of Ped., Mar., 1900.
[3] Phila. Med. Jour., July 1, 1899.
[4] Phila. Med. Jour., Sept. 2, 1899.
[5] Deut. med. Woch., No. 50, 1899; Jahrb. f. Kinderh., Bd. LI, Heft 6.

**Vaccinia.**—F. H. Fielder [1] discusses Hutchin's method of denuding the skin for vaccination by means of liquor potassæ, which plan he has found after trial to be less satisfactory than scarification. The advantages of potassium hydrate are that it is less painful and terrifying, and it does not draw blood; the disadvantages are that it takes more time and skill, it is apt to denude too large an area and cause too large vesicles, and it is less certain than scarification, because an eschar forms which interferes with absorption.

Henoch [2] states that no case of **generalized vaccinia** can be rightly considered as such unless the contents of a pustule are inoculated into an unvaccinated child or calf and produce vaccinia. D'Espine and Jeandin's case meets the requirements more nearly than any other, but the objection may be urged that it might possibly have been a case of varioloid. The majority of cases reported have not been subjected to proper criticism, and many are probably some skin disease, like eczema.

N. Vucetić [3] reports a case of generalized vaccinia in an infant 8 months old, the diagnosis from a mild attack of smallpox being made by the fact that smallpox was not present in the town at the time, nor did it develop from the case, and the development of the eruption was prolonged and irregular. The author is of the opinion that these cases are a reversion to the original type of the disease, and that the infection is carried through the body from the seat of vaccination by the blood and lymph.

J. W. Guest [4] compares vaccination **with points** to that with **glycerinized lymph** as follows:

| ADVANTAGES OF POINTS. | ADVANTAGES OF GLYCERINIZED LYMPH. |
|---|---|
| 1. The quickest and most convenient. | 1. Greater percentage in taking. |
| 2. Dries quicker, an item to office practice. | 2. Can be kept pure much longer. |
| 3. Not such profuse inflammatory reaction. | 3. Physicians are more apt to get a pure article from stores. |
| 4. Less swelling of the glands and lymphatics. | |
| 5. Less febrile action. | |
| 6. Ulcers, if resulting, not so large and destructive. | |
| 7. Patient more comfortable. | |
| 8. The physician has less criticism to bear. | |

F. Forchheimer [5] describes **vaccinoid,** and concludes that it is always modified vaccinia; that it always protects against variola, but to a less degree than vaccination; that it is usually due to faulty method and rarely to increased resistance or immunity; that in primary vaccinations it should be followed by repeated attempts, either until true vaccination is produced or until positive evidences of immunity exist; and that in the presence of variola, vaccinoid should be followed by revaccination in primary vaccinations as well as in revaccinations.

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[1] Med. Rec., vol. LVII, No. 4; Arch. of Ped., May, 1900.

[2] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.

[3] Arch. f. Kinderh., Bd. XXVIII, Hefte 5 u. 6.

[4] Pediatrics, vol. IX, No. 5.

[5] Arch. of Ped., Nov., 1899.

**Hereditary Syphilis.**—Hecker [1] says that in the **pathology of congenital syphilis** it is necessary to bear in mind the normal histology of the fetus; from a study of the organs of 113 fetuses and new-born infants, 25 of which were clearly syphilitic and 12, for comparison, were normal still-births, the author found in all the syphilitic cases a characteristic picture in the kidneys, the tissue involved being the connective tissue, the blood-vessels, and the glomeruli. In miscarriages the kidneys show a small-celled infiltration in the walls and surroundings of the smallest blood-vessels of the cortex, with sometimes a similar condition of the larger vessels of the medulla and frequently an increase of the connective tissue. In the kidneys of fetuses that have gone to full term the infiltration disappears more and more, and degenerative changes in the epithelium become prominent, although not of high degree. In infants who survive birth for a time the parenchymatous changes advance, giving the picture of a toxic nephritis, thus furnishing a probable explanation of the obscure cause of death in many syphilitic infants; that the nephritis is not due to mercury was shown by one case to which it had not been administered, the nephritis being as far advanced as in the others. In the fetus syphilis causes an increase in the proportionate weight of the kidney to that of the body, from $\frac{1}{123}$ to $\frac{1}{86}$, while in the infant the atrophic changes reduce it from the normal of $\frac{1}{94}$ to $\frac{1}{101}$. The studies on the liver have not been completed, but the author found no pathologic changes in a number of syphilitic fetuses and infants; when these changes did exist, they affected mainly the epithelium and the intralobular connective tissue, with dilation of the bile-capillaries; the normal lymph-cell aggregations in the fetal liver [see Terrien, p. 317] are not to be considered pathologic, as frequently happens. Of importance for the early diagnosis of syphilis in the living offspring of syphilitic parents is the examination of the umbilical cord; if the microscope shows no changes, syphilis in the child is not necessarily excluded, but if the examination is positive, considerable time may be gained for treatment; these changes range from a decided endarteritis or periarteritis or phlebitis to a simple round-celled infiltration of the blood-vessel walls or the surrounding tissue; simple thickening of the walls has nothing characteristic, as it is often present in normal cases.

G. Cazal [2] describes a case of obstinate hereditary syphilis in which the **manifestations** were pemphigus, orchi-epididymitis, enlargement of the superficial lymphatic glands, acute febrile pemphigus, subcutaneous gummata, ulceration of the palate, paronychia, and syphilitic pseudo-paralysis. In the treatment the author prefers inunctions, but if these are not carefully followed out, he then uses medicines by the mouth; he has not found treatment through the mother's milk to be efficacious.

**Tuberculosis.**—O. Heubner [3] treats of the **prophylaxis** against tuberculosis in childhood, in its relations to special hospitals and homes. He points out that heredity is not a necessarily present etiologic factor in every case, the great portal of infection in children, as in adults, being the air-passages and the bronchial lymph-glands; of lesser frequency are the upper and middle parts of the alimentary tract—the pharynx and small intestine. The importance of prophylaxis in childhood is given weight by the fact that many explosions of tuberculosis in adult life are but the breaking-out of latent foci which had formed in childhood. The author recommends the establishment of special hospitals for children with active tuberculosis, thus limiting to one spot—which could be selected so as to be more favorable to recovery than the children's homes—cases which would otherwise act as so many foci threatening the well. For nontuberculous but so-called scrofulous children and those who are delicate, especially the children of the poorer classes who are convalescents from the acute exanthemas and are returning to their overcrowded homes from the special hospitals, the author recommends the establishment of institutions (Heimstätten, not Heilstätten) where each child can receive special attention according to its needs, including special baths, etc., until a superabundant state of health is developed.

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[1] Jahrb. f. Kinderh., Bd. LI, Heft 3.

[2] Arch. de Méd. des Enfants, June, 1900.

[3] Jahrb. f. Kinderh., Bd. LI, Heft 1.

M. Wollenstein [1] reviews the literature of tuberculosis of the **female genital tract** in children, and reports a case in a child 2 years old, who had had measles and a persistent cough with emaciation; for several weeks before death there had been a purulent vaginal discharge. The pathologic findings were caseous bronchial glands, miliary tuberculosis of the lungs, liver, and spleen, localized tuberculous peritonitis around the rectum, and caseous tuberculosis of the right ovary and both Fallopian tubes.

A. Caillé [2] reports 13 cases of **tuberculous peritonitis** for which laparotomy was done; 3 passed from under observation at the end of a year, 2 died after no improvement and 1 after temporary bettering, the remainder being either much improved or entirely cured. The cases show the futility of medicinal treatment, the indication being for early operation. To the 3 varieties of tuberculous peritonitis ordinarily given —(1) chronic tuberculous ascites (miliary form), (2) fibrocaseous tuberculous peritonitis, (3) fibro-adhesive tuberculous peritonitis—the author adds a fourth, (4) tuberculous peritoneal tumors.

**Pertussis.**—N. R. Norton [3] reports contradictory results in the treatment of whooping-cough by injecting **carbonic acid gas** into the rectum; marked benefit followed in 143 of 150 cases treated with the nascent gas, but a second series of 20 cases, to whom the gas was given from tanks, were uninfluenced. The theory of the treatment is that it increases the amount of the carbonic acid in the blood, and therefore the interchange in the air-vesicles of the lungs must be greater and so more oxygen must be taken in. The effect of intubation on pertussis was seen in 3 cases in which laryngeal diphtheria developed; the paroxysms of coughing would be as great, but as the tube prevented laryngeal spasm, there was no whoop and the kink would end suddenly without vomiting.

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[1] Arch. of Ped., May, 1900.

[2] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.

[3] Arch. of Ped., April, 1900.

The author thinks that as the hard rubber tube does not produce any injury leading to "retained tube," the procedure would be useful in severe cases.

C. G. Kerley [1] contributes an article on **drug-values** as observed in the management of 752 cases of whooping-cough, the method of observation being to let the cases develop without medication up to the height of the paroxysmal stage, and then to try, in groups of 20, various remedies. Insufflations of resorcin with boric acid and bicarbonate of soda were found to be useless and impracticable. Inhalation with vapocresolin did no good, but medicated steam inhalations with creasote, turpentine, and ipecac were very beneficial in young and delicate children with bronchitis. Of the drugs used internally, alum, fluid extract of horse chestnut leaves, dilute nitric acid, hydrochlorate of cocain, bromoform, and belladonna were all found either to be valueless or to be very unreliable. Quinin did good in many cases, the objection being that large doses are necessary,—12 to 20 grains for children from 2 to 6 years,—and that the parents and the stomachs of the patients sometimes object to such large doses. A combination of the bromids of soda, ammonium, and potassium was of great value in lessening the severity and duration of the paroxysms, from 12 to 16 grains in 24 hours being given to a child of 1 year. Better than any of the foregoing was antipyrin, which did not depress at all; in all cases the disease was made easier in some way, either in lessening the number of the paroxysms or their severity or both. The best results were obtained with a combination of antipyrin with the bromid of soda, for a child 8 months of age ½ of a grain of antipyrin and 2 grains of bromid being given at 2-hour intervals. The use of steam in complicating bronchitis or bronchopneumonia is recommended and the importance of fresh air emphasized.

M. Heim [2] found an ointment of **antitussin** (difluorphenyl 5, vaselin 10, lanolin 85), when applied to the throat and chest, to work without fail in 7 cases in the catarrhal, and in 9 in the paroxysmal stage, which was reduced to a few days, or, at the most, 2 weeks.

K. Szegö [3] analyzed 6 cases of pertussis with reference to the effect of **sea-air** during the period of subsidence, and failed to see any abbreviation of the period; there was, however, a decidedly favorable influence exerted on the catarrhal condition of the air-passages, and the individual paroxysms were, in consequence, made milder.

Moncorvo [4] reports 26 cases of whooping-cough cured in from 3 to 8 days by means of a 1% solution of **asaprol** applied to the pharynx every 2 hours in the day.

E. Hockenjos,[5] after a review of the literature on the **cerebral affections** in the course of whooping-cough, is of the opinion that they are the result of disturbances of circulation and apoplexy, and not due to the action of a toxin or of carbonic dioxid poisoning. The severity and frequency of the paroxysms and the general bodily condition are important etiologically.

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[1] Arch. of Ped., April, 1900.

[2] Berl. klin. Woch., No. 50, 1899; Jahrb. f. Kinderh., Bd. LI, Heft 6.

[3] Arch. f. Kinderh., Bd. XXVII, Hefte 3 u. 4.

[4] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.

[5] Jahrb. f. Kinderh., Bd. LI, Heft 4.

**Influenza.**—H. Dauchez,[1] in reporting a case of influenza with cerebral manifestations, gives the differential diagnosis of **meningismus and meningitis,** the former term being introduced by Dupré to cover simple circulatory disturbances of the meninges, toxic or hysteric in origin.

T. S. Westcott [2] describes some **cerebrospinal symptoms** occurring in the course of influenza, in one case there being persistent respiratory failure for 36 hours; in the other, symptoms of meningitis being present.

N. Filatoff [3] describes a **protracted and chronic form** of influenza, of which he reports several cases in children and one in an adult. The cases originated in house-epidemics of influenza, and instead of recovery following after a week or two, the course was prolonged for from one to several months, with daily attacks of weakness, usually in the afternoon, sometimes with fever, sometimes without, usually followed by profuse sweating. Some of the cases strongly resembled malaria, and tuberculosis was also suggested, but both were excluded. Drugs seemed to be without specific effect, spermin being followed in a few cases by recovery, but having no effect in others. A change to a warm, dry climate seemed to do the most good. Prognosis as regards life is good, but the duration is uncertain.

F. Forchheimer,[4] after a consideration of the different theories suggested to explain the **dyspnea and symptoms of dilation of the heart** in influenza, reports 2 cases, and concludes that there are 2 forms of dilation, one due to the action of the toxin on the nervous system of the heart, and possibly on the myocardium, the second form occurring in such conditions in which outflow of the blood is materially interfered with on account of mechanical conditions.

**Varicella.**—A. Krjukoff [5] reports a case of **varicella gangrænosa** in an infant 14 months old. The ulcers were most numerous on the trunk, secreted a bloody pus, and were of varying size, the largest being situated on the labia majora; cultures from these gave growths of the diphtheria bacillus, either alone or with cocci; the virulence of the bacillus was tested on guinea-pigs, and found to be of high degree; in spite of antitoxin-injections, death occurred a few hours later; cultures from the pharynx were negative. In speculating on the portal of entrance for the germs, the author mentions as possibilities the hands and clothing of the patient, but inclines to the theory that the diphtheria toxin circulating in the blood and lymph found in the varicellar blebs a spot of lowered resistance, and so produced its effect. [The author evidently uses the term toxin as synonymous with bacillus.]

**Infections not Classified.**—A. Baginsky,[6] giving some of the secondary infections in children, reports a case of infection with **Bacillus pyocyaneus** (ecthyma gangrænosum); one of mixed infection with Bacillus proteus and streptococci; one of mixed infection with diplococci and streptococci; a secondary diplococcal infection in scarlatina; a bullous eruption in measles, a virulent diplococcus being obtained from the lesions; and another case of measles (tuberculosis being also present) with pemphigus, from which cultures of Streptococcus pyogenes were obtained. The cases illustrate the susceptibility of infants to infection, and the author states that they should spur us to great care in protecting infants, but should not cause a constant dread of unseen dangers.

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[1] Rev. mens. des Mal. de l'Enfance, Aug., 1899.

[2] Arch. of Ped., Jan., 1900.

[3] Arch. f. Kinderh., Bd. XVII, Hefte 1 u. 2.

[4] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.

[5] Arch. f. Kinderh., Bd. XXVII, Hefte 5 u. 6.

[6] Arch. f. Kinderh., Bd. XXVIII, Hefte 1 u. 2.

J. Pelnar [1] reports 2 cases of **pneumococcus sepsis** without pneumonia. The first was in an infant 3 months old, presenting fever and convulsions; at the autopsy gastro-enteritis, purulent meningitis of the convexity, and purulent rhinitis were found, cultures from the meningeal pus and the spleen giving the Fraenkel-Weichselbaum diplococcus. The second case was in a woman 22 years old, in the puerperium, there being an uncertain diagnosis of scarlet fever with phlegmonous tonsillitis followed by sepsis and leptomeningitis, cultures from the spleen and from the tonsillar and meningeal pus giving the pneumococcus. Pneumonia was absent in each case.

H. Audeod [2] reports a case of **meningo-encephalitis** caused by streptococci in the second week of pertussis.

Josias and Netter [3] drew from a boy with meningitis purulent fluid by lumbar puncture, and at the autopsy obtained in cultures from the cerebrospinal fluid, the meningeal pus, the heart-blood, and a renal abscess, pure growths of Staphylococcus pyogenes aureus.

T. M. Rotch [4] reports a case of **multiple osteomyelitis** in an infant 1 month old, infection with Staphylococcus aureus having probably occurred through the unhealed umbilicus. Cultures from the internal organs were negative.

W. Bloch [5] reports 18 cases of **nonsyphilitic pemphigus neonatorum,** of which he finds 2 varieties, a benign and a malignant; the former, usually fatal, being a septicemia. From 8 of these cases, studied bacteriologically, cultures of the heart-blood immediately after death gave Streptococcus pyogenes, the portal of infection not being determined. In the differential diagnosis extensive scalds, pemphigus foliaceus (Cazenave), and dermatitis exfoliativa (Ritter) must be borne in mind. The disease is not a harmless one, and as it can be transmitted from child to child by careless midwives, there should be a law compelling the reporting of cases. The benign cases may be treated by 1% salicylic acid ointment, while for the malignant cases oak-bark infusion and dry dusting-powders, zinc or talcum, are recommended.

A. M. Vargas [6] reports the case of a new-born infant presenting on the second day a general pemphigus eruption, the palms, soles, and mucous membranes being uninvolved; the fluid of the bullæ was at first clear, then becoming cloudy and slightly sanguineous, but it was germ-free. The case ran a mild course and recovered. All the customary causes of **pemphigus** being excluded, the case was considered to be toxic in origin, and of intra-uterine occurrence, the mother during the last months of pregnancy being miserably underfed, the main articles of diet being sprats, herring, and black pudding, she herself frequently having an itching eruption on the arms.

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[1] Wien. klin. Rundschau, No. 41, 1899; Jahrb. f. Kinderh., Bd. LI, Heft 6.

[2] Arch. f. Kinderh., Mar., 1900.

[3] Gaz. des Mal. inf., 1899, No. 23; Arch. of Ped., Jan., 1900.

[4] Arch. of Ped., Dec., 1899.

[5] Arch. f. Kinderh., Bd. XXVIII, Hefte 1 u. 2.

[6] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.

G. Hinsdale[1] reports the case of a new-born colored infant, seen on the second day after birth, with fever and with a fetid odor from the navel; the child cried constantly and had frequent convulsions, dying on the thirteenth day. At the autopsy, limited to the skull, there were found purulent **encephalitis,** and a cerebral abscess in the left, and a hemorrhage in the right, frontal lobe. Bacteriologic examination showed the germ to be Bacillus coli immobilis, the portal of infection being undoubtedly the umbilicus.

## DISEASES OF THE ALIMENTARY TRACT.

**Stomatitis.**—A. Epstein,[2] under the term "faule Ecken," discusses the **ulceration of the labial commissures** frequently seen in children, and for which no common term has yet been adopted. [Inasmuch as the French were the first to describe it (Lemaistre, 1886), under the term **perleche,** and as this has had a greater circulation than any other, it seems to be the best one for general adoption. The derivation is from the word *pourlècher*, to lick, children with these ulcers showing a great tendency to lick them.] Epstein gives a thorough article on the subject, pointing out the contagiousness, chronicity, and various elements in the etiology, and its benign nature except when some intercurrent acute infectious disease, like diphtheria, occurs. [We have seen the condition lead to extensive ulceration in typhoid fever, with subsequent scar formation.]

**Tonsillitis.**—The tonsils as portals of infection are discussed by E. Mayer[3] and by F. A. Packard.[4] The former concludes that infection of the system may occur through the tonsils; that tonsillar affections are frequently serious in their sequels and every step to prevent recurring attacks should be taken; that the existing disease should be actively treated, with treatment during the interim; and that after an angina the heart and other organs should be examined from time to time. Packard reports 5 cases of tonsillitis followed by organic heart disease, without articular manifestations of rheumatism having been present; nor did they follow. The author believes that either the germs gained entrance through the tonsils or their toxins produced in the tonsils were absorbed and caused endocarditis.

P. Le Damany[5] describes an epidemic of **angina** in the city of Rennes, affecting mainly boys from 10 to 18 years; the incubation period varied from 1 to 7 days. Bacteriologic examination showed the cause to be a streptococcus. Herpes was often present, and the cervical glands were swollen moderately, but remained so for some time; some cases had skin eruptions, erythemas, papules, and vesicles, and the complications were of the respiratory tract and of the upper part of the alimentary tract, the only fatal case having septicemia.

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[1] Am. Jour. Med. Sci., Sept., 1899.

[2] Jahrb. f. Kinderh., Bd. LI, Heft 3.

[3] Jour. Am. Med. Assoc., vol. XXXIII, No. 23; Arch. of Ped., Mar., 1900.

[4] Am. Jour. Med. Sci., Jan., 1900.

[5] Arch. Prov. de Méd., vol. I, No. 10; Arch. of Ped., Mar., 1900.

A. Epstein [1] refers to the rarity of **tonsillitis chronica leptothrica,** especially in children, and reports 5 cases that have come under his observation; the chronicity of the disease is well shown by the duration of his cases, which was from 2 months to a year and a half. The use of "Haller iodin-water" as a gargle 3 times a day seemed to have more influence than any other of the means employed to effect a cure.

H. Koplik [2] suggests the term "acute catarrhal lacunar amygdalitis" for all the **anginas in infants** not caused by a specific germ, as diphtheria. He tabulates the cases in infants under 1 year which were seen in 1284 cases of angina catarrhalis in children under 10 years; 25%, or 333, occurred in infants, the great majority being between the sixth and the twelfth month. The appearances of normal and diseased throats in infants are carefully described and the importance urged of inspecting the throats of all infants.

F. Huber [3] points out the important duty resting on the general practitioner to prevent serious changes from occurring or persisting in the nasopharynx in children under their care; the manifold ways in which adenoids may be harmful are detailed and the beneficial results which follow removal of the hypertrophied tissue are mentioned.

R. Fischl [4] has not found **chronic relapsing exudative angina** in childhood to be necessarily allied to or dependent on chronic hypertrophy of the tonsils. The first attack occurs usually in the second year of life, in the form of follicular tonsillitis, the recurrences appearing at intervals of varying length, sometimes every few weeks, up to puberty, when they gradually cease. A gouty diathesis and a hereditary tendency from the father seem of importance, and Fischl has observed that the attacks are more frequent and severe in houses closely connected with stables; change of residence in such instances was usually followed by improvement. A cyclic tendency seems to be of more causal influence than taking cold, and local disinfection is not of value as a preventive. Streptococci do not seem to be present so frequently as staphylococci and pneumococci. A gradual immunity does not develop as a result of the repeated attacks, the severity varying irregularly, severe attacks often following mild ones. A peculiarity sometimes seen in the course is the existence of fever for several days before the tonsils show any signs of inflammation, the morning temperature usually being normal or subnormal, with evening fever. Complications are rare. The most important preventive measure is a change of climate, prolonged residence in sea-air sometimes breaking up the tendency.

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[1] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.

[2] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.

[3] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.

[4] Jahrb. f. Kinderh., Bd. LI, Heft 3.

T. G. Brownson [1] reports an attack of tonsillitis in an infant 5 months old, with a second attack 11 months later. He refers to the rarity of the condition in infancy [which, as has been pointed out, may be in part due to its being overlooked or not looked for].

**Stomach.**—E. Pritchard [2] collects from literature 23 cases, and adds one of his own, of **hypertrophic pyloric stenosis** in infancy. From an analysis of the cases he concludes that the hypertrophy is secondary to overaction of the sphincter, and the stenosis chiefly due to spasm; that the stenosis as measured postmortem is not an accurate gage of its organic degree during life; that overaction and incoordinated contractions of the sphincter may be due to some fault in the nervous mechanism; that injudicious feeding, either quantitatively or qualitatively, may be a contributory factor of the nervous incoordination. F. E. Batten [3] contributes the report of a case with a discussion of the etiology. The different theories advanced to explain the condition are: (1) A spasmodic condition of the pylorus due to some irritant in the stomach; (2) developmental overgrowth; (3) congenital narrowing of the pylorus followed by compensatory hypertrophy of the stomach; (4) a functional disorder of the nerve-supply of the stomach and pylorus, leading to an incoordination and an antagonistic action of their muscular arrangement. The case reported was that of a male infant, breast-fed for 9 weeks, healthy at birth, but at the age of 5 weeks beginning to vomit about 5 minutes after each feeding; after 9 weeks of age the food was first cow's milk and then Nestle's food for 2 weeks, at which time the child's weight was 7¼ pounds, the abdomen was flaccid, peristaltic movements of the stomach were visible, and a firm, transverse mass could be felt in the right hypochondrium, except when peristalsis was absent; feeding by the nasal tube was instituted, and improvement began almost immediately; at the end of a month the use of the tube was abandoned, earlier attempts having led to aggravation of the vomiting; from this time on, the gain in weight was at the rate of a pound a month for 7 months, when a bronchopneumonia resulted fatally. At the autopsy the stomach measured 12 cm. in length, the total thickness of the wall of the pylorus being 5.5 mm., the circular muscular coat measuring 3 mm., the longitudinal coat 1 mm., and the mucous and submucous coats 1.5 mm. It was thought that the act of swallowing provoked such active peristalsis in the stomach that regurgitation occurred, and the case was therefore classed as belonging to the fourth group with regard to etiology.

J. H. Nicholl [4] puts on record a case of congenital hypertrophic stenosis of the pylorus, the infant, 6 weeks old, having vomited from birth and being greatly emaciated. Loreta's operation was done, the pylorus being dilated until the peritoneal coat was ruptured; recovery was complete.

J. Finlayson [5] reports a case of **sarcoma of the stomach** in a child 3½ years old who, during life, presented diarrhea alternating with constipation, occasional attacks of vomiting, and a progressive anemia, without demonstrable organic change, the tumor being found at autopsy to involve the posterior wall.

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[1] Arch. of Ped., Aug., 1889.

[2] Arch. of Ped., April, 1900.

[3] Lancet, Dec. 2, 1899.

[4] Glasgow Med. Jour., April, 1900.

[5] Brit. Med. Jour., Dec. 2, 1889.

**Intestinal Tract.**—H. Koplik,[1] in the President's address to the American Pediatric Society, discusses the ambulatory and hospital management of the **gastro-intestinal derangements** of infancy in the summer months among the poor of large cities. After referring to the mortality which these disorders cause, to the great need for clean milk, and to some points connected with infant feeding, the author discusses the advantages of dispensary as compared with ward treatment. He makes the suggestion that the best solution of the problem will be the establishment in mountainous regions of camps, huts, or cottages arranged over acres of land around a central administration building, thus avoiding the crowding together of a number of sick children, with the attendant disadvantages.

C. de Lange [2] gives the results of a series of careful studies on the normal and pathologic **histology** of the intestinal canals of children.

H. Finkelstein [3] treats of **sepsis** in early childhood and its relationship to the intestine, concluding as follows: Severe sepsis, characterized by intense general poisoning, may run its course with gastro-enteric symptoms, sometimes becoming cholera-like; bacteriologic study of the blood does not show that sepsis finds its single expression in a gastroenteritis, although certain rare cases of acute characteristic septicemia occur with an enormous number of bacteria in the blood. The complications in intestinal diseases involving the organs are clearly ascending or descending processes which depend on the intestinal disorder only to the extent of the latter producing a predisposition; sometimes one of these complications may cause secondarily an equally important septic general infection. Secondary septic as well as agonal infection occurs almost without exception from the skin or a mucous membrane other than the intestine, the colon bacteria furnishing no evidence of the source, as bacteria can penetrate the intestinal mucosa only when it is deeply ulcerated; a streptococcal infection is thus possible from a streptococcus-enteritis. Local or general septic processes are to be looked on rather as complications than as causes of chronic atrophy and allied conditions.

Avirагnet [4] reports a number of cases of gastro-intestinal indigestion dependent on chronic **rhinopharyngitis or tonsillitis,** and incurable by the usual measures as long as the throat condition was unchecked.

Escherich [5] reports 3 epidemics of **catarrhal dysentery** in children, comprising 40 cases in all, the stools giving pure cultures of the colon bacilli, the blood-serum in each case causing agglutination in cultures of the bacilli from the same case; the author concludes that the colon bacillus was the cause of the epidemics, and believes that he carried the infection to his own child, who was one of the patients.

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[1] Arch. of Ped., May, 1900.

[2] Jahrb. f. Kinderh., Bd. LI, Heft 6.

[3] Ibid., Heft 2.

[4] Arch. de Méd. des Enfants, Feb., 1900.

[5] Centralbl. f. Bakt., XXVI, 13, 1899.

Mellin [1] investigated the virulence of the different **varieties of Bacterium coli commune** isolated from the stools of two children with gastro-enteritis and of one healthy child; 22 varieties were obtained, differing greatly in virulence, which did not seem to be connected with the severity of the disease in the patient from whom the germs were obtained. The author ascribes this to the different culture-mediums upon which the germs were grown, and is of the opinion that an intestinal mucous membrane, irritated either thermochemically or mechanically, probably furnishes a favorable soil for the germs.

P. Nobécourt [2] studied the **streptococci** found in the intestines of sick and well infants, and found that there were no characteristics of morphology or virulence by which the origin of the germs, whether from the sick or from the well, could be told.

The rôle of **microbes in the gastro-enteritis of infants** is the subject of an extensive analysis, with a thorough review of the literature, by A. B. Marfan.[3] After giving the historic development of the subject and the diverse opinions of the different investigators, of whom the author is one, he seeks to point out what is certain knowledge, what is probable, and what is improbable. The bacteriologic examination should include the following procedures: (1) Microscopic examination of fecal matter; (2) cultures from stools; (3) study of the virulence of the microbes isolated, and an attempt to reproduce the disease experimentally, avoiding here the danger of forming conclusions too hastily from what occurs after subcutaneous, intraperitoneal, or intravascular injection as to what occurs after ingestion by the mouth; (4) study of the serum reaction; (5) study of the products (toxins) elaborated by the intestinal microbes; (6) experiments in general infection of the organism by the intestinal microbes. The bacteria studied successively are: (1) The colon group: (2) streptococci; (3) staphylococci; (4) Bacillus pyocyaneus; (5) Proteus vulgaris; (6) proteolytic bacteria, or the ferments of casein; (7) Bacillus enteritidis sporogenes; (8) yeasts; (9) protozoa; (10) microbic associations. As a result of this study, it is seen that in gastro-enteritis the infection may be primary, ectogenous, in which case it is the essential cause of the disease; or it may be secondary, endogenous, occurring as an episode. The ectogenous infections actually known are streptococci, especially the variety described by Escherich, and Bacillus pyocyaneus; perhaps also Proteus vulgaris and the colon bacillus; these ectogenous infections are contagious, and may assume an epidemic character. The agents of endogenous infection are the constant inhabitants of the intestine; those whose action seems to be established are the colon group, the proteolytic bacteria, and perhaps also Proteus vulgaris. Although bacteriology has shown that the rôle of infection is important in these affections, nevertheless it is sometimes secondary to other elements. Combining the facts of bacteriology, pathology, and clinical observation, the local and general manifestations of gastro-enteritis form

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[1] Arch. f. Kinderh., Bd. XXVIII, Heft 1.

[2] Jour. de Phys. et de Path. Gen., Nov. 15, 1899.

[3] Rev. mens. des Mal. de l'Enfance, Aug.–Nov., 1899.

4 groups: (1) The faulty elaboration of food, properly designated as *dyspepsia*. (2) *Infection* of the intestinal contents, due either to exaltation in virulence of the bacteria habitually present (endogenous infection), or to the accidental entrance of pathogenic germs (ectogenous infection). (3) "*Toxicity*" of the gastro-intestinal contents, which occurs either by the introduction through the buccal passages of poisons from without (ectogenous intoxication); or from the elaboration of toxins by pathogenic microbes, more or less specific, accidentally introduced into the alimentary tract (specific endogenous intoxication); or, finally, from ordinary dyspeptic fermentations set up by the germs always present (common or dyspeptic endogenous intoxication), consisting in the transformation of lactose to acid substances and sometimes of nitrogenous material to indol, skatol, etc., probably with the production of toxins. (4) Modifications of the *gastro-intestinal wall*, as shown by disturbances of secretion, peristalsis, tonicity, and sensibility (anorexia, vomiting, diarrhea, meteorism, pain). A little reflection is only necessary to see that when one of these elements is present, the others will develop in time, so that they will then be associated in different proportions. Nevertheless, an etiologic classification based on the foregoing is feasible, for it is often possible to say of one case of gastro-enteritis, this is dyspeptic in origin; of another, this is an infection primarily; of another, this started as a toxic gastro-enteritis. There is still another form, the secondary gastro-enteritis, which occurs in different diseases, such as measles, pneumonia, etc., and the way in which it arises varies; it may be due to an elimination of the germs or toxins in the bile, or the digestive powers may be weakened, or, as in bronchopneumonia, the septic material may be swallowed. It is thus seen that the complexity of pathogenesis renders it impossible to assign to a fixed clinical form of gastro-enteritis a primary cause always the same, nor will the same primary cause—*e. g.*, streptococci—always give the same clinical form of gastro-enteritis, because the preceding state of the alimentary tract, the characteristics of the intestinal flora, and the vital resistance of the patient will direct the action of the same primary cause in very different ways.

J. L. Morse [1] reviews the literature on the **renal complications** of the acute enteric diseases of infancy, finding great variations of opinion among writers, the majority, however, stating that renal complications are frequent in acute enteritis, while few base their opinions on observations. The author seeks to explain the contradictions in pathologic findings by different investigators as being due to difference in the clinical material, those who found nephritis present probably studying very acute cases of cholera infantum, while those who did not find nephritis common had more varied cases. In 70 cases of fermental diarrhea and ileocolitis, the author examined the urine obtained by catheterization,—once only in the majority of cases,—and found albumin present in but 11, and this was considered to be the result, not of inflammation, but of degenerative changes due to bacteria or toxins. The author therefore concludes that nephritis is a very unusual complication of the acute diarrheal diseases of infancy, and that the occurrence of albumin and renal elements in the urine is not of bad prognostic import. None of the cases were cholera infantum.

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[1] Arch. of Ped., Sept., 1899.

D'Orlandi [1] examined the blood of 19 infants with enteritis, and found no striking or characteristic change; the red cells were reduced and the white cells were occasionally increased, but usually slightly decreased, the proportions of the mononuclears, lymphocytes, and eosinophiles being raised, the significance, if any, of which is not understood.

J. Comby,[2] basing his remarks on 15 cases, discusses the **anemia of dyspeptic infants,** in its etiology, symptoms, prophylaxis, and treatment; he evidently does not consider von Jaksch's pseudoleukemic anemia of infants to be an essential blood disease, but a part of a chronic gastro-enteritis. In the outline of treatment milk is given a minor place in the diet, and he recommends the giving of not more than half a liter daily.

Spiegelberg [3] made bacteriologic and histologic studies on the relation between the **pneumonias** which so frequently complicate enteritis in children and the intestinal infection. The bacteria in the intestine grow especially in the superficial layers of the mucous membrane, and to enable these to penetrate into the blood or lymphatic circulation, ulceration of the intestinal wall and invasion of it by the bacteria are necessary; but this is actually a rare occurrence. Study of the lungs leads to the conclusion that the pneumonias are bronchogenic, their development being favored by the effects of the intestinal disease.

Poix [4] reports good results in a limited number of cases in the treatment of infantile diarrhea with the use of **dermatol** (bismuth subgallate) and a diet of sterilized water. Instead of milk or any other food, the water is given for the purpose of preventing intestinal fermentation, of allowing rest to the stomach and intestines, of avoiding drying of the tissues, and of maintaining the flow of urine.

F. P. Sunico [5] reports cases successfully treated on similar lines, no food, but only water, being given for periods ranging from 1 to 5 days; other measures were used, such as intestinal antiseptics.

**Appendicitis.**—Goyens [6] has the distinction of reporting an attack of appendicitis in probably the **youngest patient on record,** an infant 6 weeks old, the autopsy showing a general peritonitis with a gangrenous, perforated appendix; the cause was bottle-feeding setting up an infectious gastro-enteritis.

**Tympanites.**—Leo [7] gives as the causes for tympanites in children weakness of the intestinal and abdominal walls and the increase of gas. Dyspeptic children may be grouped into 3 classes: those in whom no gas has resulted from fermentation, the composition of the "stomach air" being, as in healthy children, that of swallowed air; those in whom the same gases are present, but, as the result of yeast-fermentation, the amount of $CO_2$ may be increased up to 17%; those in whom H up to 32.66% and marsh-gas up to 9.48% are present, the latter being favored by atony of the stomach and stagnation of the contents, which lead to butyric acid fermentation.

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[1] Rev. mens. des Mal. de l'Enfance, vol. XVII, 1899.
[2] Arch. de Méd. des Enfants, June, 1900.
[3] Arch. f. Kinderh., Bd. XXVII, Hefte 5 u. 6.
[4] Rev. mens. des Mal. de l'Enfance, Jan., 1900.
[5] Arch. de. Méd. des Enfants, April, 1900.
[6] Gaz. méd. Belge, vol. XII, No. 14; Arch. of Ped., Mar., 1900.
[7] Arch. f. Kinderh., Bd. XXVIII, Hefte 1 u. 2.

**Digestion of Starches.**—F. Callomon,[1] using as a guide Schmidt's work on the fermentation of expelled feces and its relation to digestion, found that similar conclusions could not be drawn from a study of the stools of infants. Schmidt has found that there were 2 varieties of fermentation: one, early, within 24 hours after expulsion, in which the carbohydrates were broken up and $CO_2$ set free; the other, late, in the second 24 hours, giving rise to H, carbureted H, a small amount of $CO_2$, and putrefactive products. The early fermentation is an indication of the digestion and absorption of starches in the intestine. Callomon found that this did not hold for breast-fed infants who were gaining regularly and whose digestion was normal.

**Intussusception.**—The treatment of intussusception was discussed by the Section on Pediatrics of the New York Academy of Medicine.[2] Papers introducing the subject were read by F. Kammerer, on "The Mortality and Treatment," and C. L. Gibson, on "The Necessary Factors in the Successful Treatment." Kammerer refers to the obscure etiology, the easy diagnosis, and the present tendency to an earlier resort to operative interference than formerly. The only bloodless method worthy of serious consideration is the injection of fluids into the rectum, and this is applicable only to cases of intussusception affecting the large intestine. Only in chronic cases may enemas be employed with impunity, and that many weeks after the onset, while in acute cases there has been fixed arbitrarily a limit of 24 hours after the onset, later than which reduction by enema may be dangerous; even before the end of 24 hours serious changes may have taken place in the neck of the intussusceptum. The most serious objection, however, is inability to recognize whether or not reduction has really been accomplished. The following rules are laid down for the use of enemas: (1) In acute cases an attempt at reduction should be made only very early in the case, and once only; (2) this attempt should always be made under complete anesthesia, with relaxed abdominal walls; (3) hot water should be employed in preference to iced water, although the latter is said to have effected reduction when the former failed; (4) in very acute cases the method should not be employed; (5) after one failure, laparotomy is indicated. The mortality after laparotomy depends largely upon whether or not reduction is possible; irreducible cases have a mortality double that of reducible, and the question of how to treat an irreducible intussusception is still open, the author apparently favoring resection of the intussusceptum as giving better results. Laparotomy by the author in infants 6 and 3 months old was well borne, and other operations on infants are cited to show that they, as a class, are not so much shocked by major operations as are adults.

[1] Jahrb. f. Kinderh., Bd. L, 1899.

[2] Arch. of Ped., Feb., 1900.

Gibson, in his paper, discussed the influence of the duration of obstruction on the prognosis, stating that whether the intussusception is reducible, irreducible, or gangrenous depends almost entirely on the duration of the obstruction, the mortality of laparotomies for intussusception rising from 36% in the reducible to 64% in the irreducible and to 95% in the gangrenous. In the discussion on the subject, F. H. Wiggin said that the manipulation in the injection method was more severe on the child than operation, a study of the subject convincing him that the injection treatment was not only unsafe, but totally unreliable, the results from laparotomy by skilful surgeons being better. W. Meyer also urged surgical treatment for the condition, adding that the variety of the majority of cases (ileocecal), and the traction necessary in laparotomy to reduce the intussusception, negatived the idea that much could be accomplished by injections. A. Jacobi was of the opinion that the enema treatment was useful if carried out on a certain definite plan, which was to raise the syringe not higher than from 12 to 18 inches above the anus of the patient, whose head should be down and hips up; chloroform anesthesia should be used, and while warm water is flowing in, the abdomen should be gently manipulated, the anus being held tightly closed; if the first attempt is a failure, the injection should be repeated every 1 or 2 hours for 2 or 3 times, and if still unsuccessful, operation should be performed at once. C. G. Kerley, referring to diagnosis, said that many cases of bloody stools with slight fever and marked prostration were probably intussusception; he referred to 2 cases which he had successfully treated by injection, and he was of the opinion that it should be used only in the early stage.

Alfred Hand, Jr.,[1] has reported a case of intussusception in an infant 4 months old, the tumor being felt in the line of the descending colon; for a day the stools had been only bloody mucus, but 5 hours after reduction by enema, the syringe being elevated 2 feet, fecal matter reappeared; opium seemed to be of great value in the treatment in preventing recurrence, which was at one time threatened.

**Dilation of the Colon.**—L. Concetti[2] treats of certain **congenital malformations of the colon** in children causing habitual **constipation,** eliminating from the discussion all cases of constipation dependent on diet, drugs, diseases of other organs, such as the liver, or of the nervous system, and all congenital or acquired defects in the lumen of the intestine, such as imperforate rectum, tumors, fecal accumulation, strictures, etc. He gives the essential points in the histories of 28 cases collected from the literature, and adds in detail 2 cases of his own, of congenital dilation of the colon. To reverse his order, the second case was that of a boy, 8 years old, whose bowels had not moved for exactly 1 month; the abdomen had always been distended and there was rarely a movement oftener than every 2 or 3 days, there being several prolonged periods of absence of movement, meconium being voided first 15 days after birth, with similar periods at 2, 4, and 6 years of age. When first seen, at the end of the month of obstipation, over 10 kg. of feces were scooped out of the rectum in 3 days; in 2 weeks of medicinal treatment marked improvement followed. The first case was in a girl 2½ years old, who had periods of obstipation ranging from 5 to 15 days; the abdomen was greatly distended, measuring 69 cm. in circumference; for a time it was possible to obtain regular evacuations by medicinal means, but these soon failed, the obstipation being shortly followed by vomiting, emaciation, and a watery diarrhea, death occurring in collapse. Autopsy with microscopic examination gave the following findings: Congenital aplasia of the muscularis of the colon, just above the rectum; dilation of the sigmoid flexure; dilation and hypertrophy of the entire colon, with ulceration and perforation. The author, from an analysis of all the cases reported, finds 3 main anatomic types: (1) Simple excessive length of the colon ("makrocolie"; *makros*, long); (2) a great uniform increase in the internal diameter of the colon, with thickening of the walls ("megalocolie"; *megas*, great); (3) simple congenital dilation of a more or less extensive section of the colon, with or without compensatory hypertrophy and dilation of the adjacent section ("ectacolie"; *ektatos*, dilated). In the first group the long descending colon and sigmoid flexure, having little room in the small pelvis of the child, must make several sharp bends, leading to slowing of the onward progress of the feces, with accumulation, and to all the general disturbances of nutrition which are set up by the absorption of toxic, putrefactive substances; the fecal mass may also irritate the intestine and cause an offensive, watery diarrhea. The prognosis of these cases, under rational dietetic treatment with massage, is good; for as the child grows, the disproportion in the length of the colon lessens and the abdominal and pelvic cavities become more roomy. In the second group are those cases in which at birth the colon is larger than normal both in its cavity and in the thickness of its walls, the latter being due to an increase in the embryonic connective-tissue elements; these hinder the activity of the muscularis and cause it first to hypertrophy and then to weaken, so that the dilation increases, with all the secondary results; the new connective tissue also involves the small arteries, interfering with the nutrition of the mucosa, and the feces therefore easily irritate it, producing ulceration. The chance for life is much less than in the first group, although if the arteries are not extensively involved and the mucosa maintains its nutrition and prevents the absorption of toxic products, life may be prolonged, as in Formad's case, to 21 years, or even to 50 years, as in Bastianelli's case. The author places his second case in this group. The third group is typified by the author's first case; the condition at birth is merely a deficiency in the muscularis at some part of the colon; as the feces reach this section there is no power to propel them, and they stagnate, producing dilation of the section; then there occur hypertrophy and dilation of the colon above, to a degree usually depending on the length of life, which is usually not more than a few years, the fecal accumulation causing its customary sequels, ulceration, toxemia, etc., death resulting often from an acute colitis. Except in the first group, medicinal treatment fails, so surgical measures should be adopted early.

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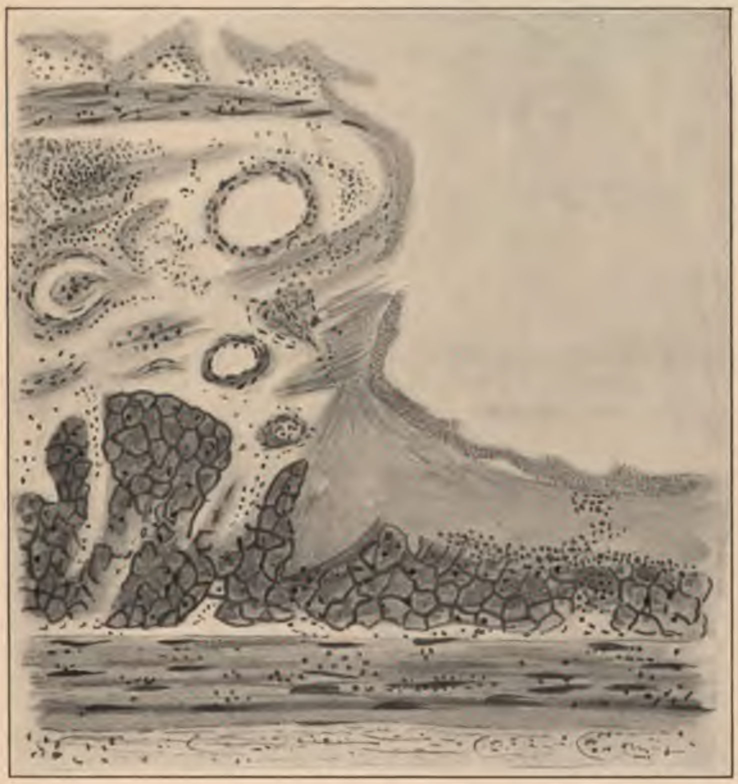
[1] Arch. of Ped., Aug., 1900.

[2] Arch. f. Kinderh., Bd. XXVII, Hefte 5 u. 6.

PLATE I.



Habitual constipation causing malformation of the colon. Illustration shows hypertrophy of transverse muscular coat, infiltration of areolar tissue, degeneration of the mucosa; to the right, destruction of tissue down to the muscular coat (Concetti, in Arch. f. Kinderheilk., Bd. XXVII, Hefte 5 u. 6).

J. P. C. Griffith[1] reports a case of congenital idiopathic dilation of the colon in a boy 3 years old, who had been constipated from birth, with distention of the abdomen from 5 months of age. On admission to the hospital, diarrhea was present, but the tympany was so excessive that laparotomy was necessary to form an artificial anus; the child was in such wretched general condition that he did not survive; only a partial autopsy was allowed, the colon being found tremendously dilated, while the ileum was contracted just above the cecum. The author collects 23 cases from the literature, which he considers are idiopathic, showing that boys are more often affected than girls, in the proportion of 5 to 1. The most prominent symptoms are abdominal distention and obstinate constipation, with, at times, attacks of diarrhea, the last, if persistent, being an unfavorable sign; vomiting is rare, but the distention often causes dyspnea. Of the patients, 18 of the 24 died in childhood, but only 3 are positively known to have lived to adult life. Ulceration of the mucosa was found in all the cases with severe diarrhea. The treatment should be directed to the chronic constipation, and if relief is not prompt, surgical measures should be adopted.

A. Johannessen[2] reports 3 cases, one dying at the age of 30 months, the others still living at the ages of 14 months and of 3 years; the author reviews the literature bearing on the etiology, and takes the ground that the condition is the result of excessive length of the sigmoid, which is kinked. F. T. Stewart and Alfred Hand, Jr.,[3] report a case in a boy 6 years old, terminating fatally.

**Liver.**—Moncorvo[4] reports a case of **abscess of the liver** occurring in a boy and following traumatism.

E. Terrien,[5] after examining a number of fetal livers with reference to the collections of small round-cells (**embryonic infiltration**) and their disappearance, found that they normally disappeared in the first few days after birth, a persistence being pathologic, and that they are recognizable from the infiltrations which occur in gastro-enteritis—the so-called false fetal masses. The number of observations was too small to determine the significance of the embryonic infiltrations. In children with gastro-enteritis[6] there may be these infiltrations, which are probably intravascular, other changes being seen in the connective-tissue and the parenchyma (fat); the author describes the lesions of the liver which may occur in gastro-enteritis, detailing the steps leading up to cirrhosis.

R. G. Freeman[7] describes a number of **lesions** of the liver found at autopsy. His summary is as follows: (1) Descent of the liver down the right side of the abdomen, so that the right lobe reaches below the crest of the ilium, occurs not very rarely in infants, and particularly in those in whom the liver is enlarged. (2) Fatty livers occur very frequently in the infants and children which die at the Foundling Hospital, or in about 41% of all cases. (3) The condition of nutrition of the child, as expressed by the absence of fat in general and wasting of tissue, apparently has no connection with the fatty condition of the liver, the condition of nutrition in the cases having fatty livers averaging about the same as in the whole number of cases. (4) Fatty livers occur rarely in the following chronic wasting diseases: Marasmus, malnutrition, rachitis, and syphilis, unless such condition be complicated by an acute disease. (5) With tuberculosis fatty livers occur not more often than with other conditions. (6) Fatty livers occur most often with the acute infectious diseases and gastro-intestinal disorders. (7) The two cases of cirrhosis of the liver examined by the writer ran a comparatively acute course. On section the livers showed a marked hyperplasia of the so-called new-formed bile-ducts. (8) Focal necrosis of the liver may be a lesion of measles.

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[1] Am. Jour. Med. Sci., Sept., 1899.

[2] Rev. mens. des Mal. de l'Enfance, Feb., 1900.

[3] Arch. of Ped., Mar., 1900.

[4] Rev. méd., Sept. 19, 1899; Pediatrics, vol. VIII, No. 12.

[5] Rev. mens. des Mal. de l'Enfance, Oct., 1899.

[6] Rev. mens. des Mal. de l'Enfance, Oct., 1899.

[7] Arch. of Ped., Feb., 1900.

**Diaphragmatic Hernia.**—Cases of congenital diaphragmatic hernia are reported by S. W. Kelley [1] and by I. A. Abt.[2] In both cases there was a deficiency of the left part of the diaphragm, so that the left lobe of the liver, the stomach, the duodenum, the small intestine and a part of the colon were in the left half of the thorax; cyanosis was intense at birth and resuscitation was impossible. Abt's case also presented a congenital goiter.

**Intestinal Parasites.**—G. F. Still [3] urges, in the treatment of threadworms, a large injection—12 to 18 ounces or more—in order to reach the real habitat of the parasites, which is not the rectum, as is popularly supposed, but the cecum and vermiform appendix; even large injections sometimes fail, so the use of santonin by the mouth is recommended. The author holds that it is possible for the ova to be hatched in the appendix without first being swallowed, and they may sometimes set up a catarrhal condition of the appendix simulating appendicitis. It may be added, in view of the case reported by C. H. Frazier,[4] that the oxyuris is a cause of appendicitis in some instances.

## DISEASES OF THE CIRCULATORY SYSTEM AND BLOOD.

Von Starck [5] quotes the opinion of Hochsinger and others, who have stated that **cardiac murmurs** in the first 3 years of life are always organic, and while in his experience accidental murmurs are very rare, yet he has seen 4 cases, which he reports, in which the murmurs were heard repeatedly, the autopsies revealing nothing abnormal in the hearts. The author also discusses the diagnosis of congenital heart lesions.

C. W. Townsend,[6] after a study of 30 cases of **congenital heart disease,** arrived at the following conclusions: (1) Duration of life: Of 19 cases kept under observation, 13 have died at an average of 3 years, the youngest being 3 hours, the oldest 9½ years; of 6 living cases, the youngest is 4 months, the oldest is 28 years. (2) Cyanosis: This was present in all cases with one exception. (3) Cardiac signs: These were absent in one-third of the cases, 2 of these coming to autopsy and showing marked cardiac malformation. In two-thirds murmurs were present, generally diffuse and systolic in time, with or without a thrill, and loudest at either the base or the apex. (4) Blood count: The number of red corpuscles in the blood was increased in all but 1 of 14 cases examined, as is found to be the case in cyanosis from other causes. The exception occurred in the only case devoid of cyanosis. The average red blood count in 13 cases was 7,573,585 cells per cubic millimeter.

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[1] Arch. of Ped., Aug., 1899.

[2] Ibid., April, 1900.

[3] Brit. Med. Jour., No. 1998, 1899; Arch. of Ped., Aug., 1899.

[4] Univ. Med. Mag., Mar., 1900.

[5] Arch. f. Kinderh., Bd. XXVIII, Hefte 3 u. 4.

[6] Arch. of Ped., Sept., 1899.

J. P. C. Griffith [1] reports the case of an 8-months-old boy who had experienced attacks of cyanosis from the age of 5 months; death followed an attack of pneumonia, and the autopsy showed a transposition of the viscera and of the great vessels, with pulmonary stenosis and perforate septum ventriculorum, but without a true dextrocardia, as the auriculoventricular valve on the left was composed of 2 leaflets, while that on the right had 3.

L. Vervaeck [2] reports a **congenital malformation** of the heart, consisting of deficient auricular and ventricular septa, absence of the tricuspid orifice and valves, malformation of the aortic and pulmonic valves, and incomplete development of the right ventricle and pulmonary artery; death occurred at 4½ years, a murmur having been present throughout life, but cyanosis developing only shortly before death. The author describes the course of the blood and discusses the production of the anomalies, and also the question of the cause of cyanosis in congenital heart disease, explaining its absence in this case, in which there was pulmonary stenosis with a mixture of arterial and venous blood, by hyperoxygenation of those red blood-cells which passed through the lungs. Vervaeck [3] nevertheless thinks that the most plausible explanation of **cyanosis** is the admixture of arterial and venous blood, and that the influence of pulmonary stenosis can not be invoked as a cause, inasmuch as in 50% of the cases of cyanosis pulmonary stenosis is absent, and it is often present without causing cyanosis.

A. C. Cotton [4] reports a case of **pulmonary stenosis and infective endocarditis** in a boy 11 years old, noma starting several weeks before death. The autopsy showed a congenital malformation of the heart, consisting of stenosis of the pulmonary conus, malformation of the pulmonary valve (2 cusps), and perforate ventricular septum; in addition, there were acute mural endocarditis and an aneurysm of the right ventricular wall, streptococci being obtained in cultures from the vegetations in the heart. The author also [5] observed a new-born infant, dying on the fourth day, having passed only 1 cc. of urine during life; the autopsy revealed a defective septum between the pulmonary artery and the aorta, a patent ductus arteriosus and foramen ovale, and general infection with Bacillus mucosus capsulatus.

T. Escherich [6] gives the notes of a prematurely born infant, cyanotic from birth, with a loud systolic murmur at the base of the heart; death occurred 12 hours after birth, and the autopsy showed a **pneumonia and a patulous ductus arteriosus,** the heart being normal; the pneumonia, of intra-uterine origin, doubtless took part in keeping the ductus open; and the author suggests that Schultze's method of artificial respiration would have been beneficial. The literature on patulous ductus arteriosus is reviewed.

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[1] Arch. of Ped., Aug., 1899.

[2] Arch. de Méd. des Enfants, June, 1900.

[3] Jour. méd. de Bruxelles, Feb. 1, 1900; Arch. de Méd. des Enfants, June, 1900.

[4] Arch. of Ped., Dec., 1899.

[5] Ibid., Oct., 1899.

[6] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.

L. E. Holt [1] reports a case of **cardiac malformation** in a child 19 months old, the symptoms during life being poor development, with occasional attacks of cyanosis; examination showed slight enlargement of the heart to the left, with a systolic murmur over the whole left chest, of maximum intensity midway between the nipple and the median line, and at times there was a double murmur at the apex; there was marked epigastric pulsation and the aortic second sound was intensified; posteriorly a systolic murmur was heard over both sides, but loudest on the left near the spine at the level of the sixth to the eighth dorsal vertebra. Bronchopneumonia, cyanosis, and edema of the face and feet developed, but all except the consolidation of the lung disappeared 2 weeks before death, which resulted from asthenia. At the autopsy the much enlarged aorta was found to communicate with both ventricles, the upper portion of the ventricular septum being deficient; there was complete atresia of the pulmonary orifice with great reduction in the size of the pulmonary artery, the ductus arteriosus being absent; the aortic, mitral, and tricuspid valves were normal; the left common carotid artery arose before the innominate and the aorta gave off, from the lower part of the descending arch, a large branch ¼ of an inch in diameter, which passed in front of the aorta, communicating with the trunk of the pulmonary artery and giving off 3 branches to each lung. There was a general tuberculosis. The absence of cyanosis for the greater part of life is noteworthy, and the case illustrates well the contention that cyanosis does not depend on admixture of arterial and venous blood.

J. W. Troitsky [1] gives an exhaustive review of the literature on the subject of the normal limits of the superficial and deep areas of **cardiac dullness** in childhood; conclusions are drawn from these writings and from his own studies on 229 healthy children. The details of the studies are too extensive to condense in a brief abstract.

W. P. Northrup [1] contributes a brief note referring to the fact that a case of acute simple **endocarditis** has never been observed in the New York Foundling Hospital in the past 27 years, the service covering the admission of 27,000 infants, with autopsy in between 2000 and 3000. Heart-murmurs have been frequently noticed, but they have been transitory or unexplained at the autopsy. One case of malignant endocarditis has been seen as a part of a septicemia.

S. S. Adams [1] has collected from literature 47 cases, including his own, of septic endocarditis in children under 14 years of age. The diagnosis rests mainly on the presence of irregular fever, the changing character of the cardiac murmur, and its tendency to form emboli, but examination of the blood and the cultivation of micro-organisms in nutrient media are of great help. H. Hun [1] details an attack of primary simple acute endocarditis in a girl 13 years old, resulting in mitral insufficiency and stenosis; during the course neither pneumonia nor rheumatism was present, although the first was suggested by a few rales and herpes, and the second by the family history and by a few nodules on the fingers.

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[1] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.

B. Rogers [2] reports an **aneurysm of the aorta** in a girl 10 years old, who died suddenly after an illness of 6 months, during which there had been a murmur, heard best over the aortic and sometimes over the pulmonic cartilage. The opening of the aneurysm was about ⅔ of an inch long and was ½ of an inch above the anterior semilunar valve of the aorta.

Rosenstein [3] reports a very unusual case of **chronic tuberculous myocarditis** with beginning aneurysmal formation in the wall of the left ventricle. The patient was a boy 11 years old, with tuberculous lesions of the hip-joint, lung, and kidney, the adherent pericardium being probably an old tuberculous lesion.

E. Weill and L. Gallavardin [4] report a case of acute **symphysis of the pericardium** in a girl 12 years old, with fatty degeneration of the myocardium, kidneys, and liver; rheumatism was evidently the cause. In a consideration of the subject the authors find that rheumatic pericarditis has a mortality of about 50%.

E. Weill [5] gives a valuable and interesting discussion of the treatment of **infantile cardiopathies,** to which justice can not be done in a short abstract. Among the salient points he shows that the hopefulness of therapeutics is much greater when the patient is a child than when he is an adult, the chief reasons for which are the anatomic and physiologic differences in the circulatory apparatus in early from those in middle life; stress is laid on the prevention of rheumatic endopericarditis by the thorough use of the salicylates, and reply is made to those who claim that the use of the salicylates predisposes to cardiac involvement by referring to the frequency with which the slight manifestations of rheumatism in children are followed by heart disease, the treatment having been very short or in many cases omitted altogether. Chorea, depending on a rheumatic soil, is an indication for the salicylates, which, although they possess no action on the nervous disease, may yet protect the heart. In the treatment of an actual lesion in the acute stage the importance of mental rest is urged as well as rest of the body. When a valve has become inflamed, the possibility of absorption of the products of inflammation are much greater in children because the blood-vessels extend further up in the valves than at a later period of life; and even if one valve is permanently damaged, the other valves at the same opening may in time stretch so as to produce a perfect compensation and a practical cure. Pericarditis always makes the outlook dark. The discussion of the treatment of the period of compensation is especially to be commended for its detail and good sense.

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[1] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.

[2] Ped., vol. VIII, No. 4.

[3] Zeit. f. klin. Med., Bd. XXXIX, Hefte 1 u. 2.

[4] Arch. de Méd. des Enfants, April, 1900.

[5] Arch. de Méd. des Enfants, Jan. and Feb., 1900.

C. W. Chapman [1] gives excellent advice on the **treatment** of heart disease in childhood and youth, pointing out not only the uselessness, but the risk, of giving digitalis to patients with murmurs when the heart is acting well, as the increased force to the contraction of the right ventricle might lead to rupture of pulmonary capillaries and dilation of the left auricle and also of the right ventricle. When the heart begins to dilate, then the digitalis group is not only of value, but is a necessary part of treatment, and it is well to precede digitalis by free purgation. If large doses are necessary, the recumbent posture is the best. Venesection or leeches may be resorted to if cyanosis comes on; mercury is frequently indicated to unload the liver, and nux vomica is valuable in small doses with interruptions; alcohol may be needed, but is usually harmful; belladonna externally relieves cardiac pain. Nauheim baths are rarely indicated in children, but exercises do good in properly selected cases.

L. E. Holt [2] lays emphasis on the importance of **prolonged rest in bed** after acute cardiac inflammations in children, measuring the time not by several weeks, but by as many months.

F. J. Poynton [3] reports the result of a study of **cardiac overstrain** in the young. He outlined the areas of deep cardiac dullness in 9 youths before they entered on a period of training in athletics lasting 2 months; on the day after the races he was able to examine them and to obtain tracings to compare with the first set. Tachycardia was not present in any boy, but, as a rule, the heart was found enlarged to the right and, on the left, upward in the region of the infundibulum of the right ventricle. One boy at the first examination was found to have 2 systolic murmurs—one at the apex, the other at the base; at the second examination the murmur at the apex was unchanged, while the other had disappeared. The author discusses the different games and sports with regard to their "exertion value," and urges a thorough knowledge of them on the part of medical men, in order that directions with regard to them may be consistent. The case is cited of a boy with a weak heart who was forbidden to play cricket but allowed to play racquets, the physician who gave the advice evidently believing that racquets and tennis involved no great exertion.

**Diseases of the Blood.**—J. G. Kinneman [4] reports the case of a new-born infant in whom **hemorrhage** occurred from the cord and around the umbilicus at birth, blood being discharged from the bowel 12 hours later and death occurring 36 hours after birth. There was no family history of tuberculosis, syphilis, or hemorrhagic diathesis; 4 other male infants had died at birth—one from hemorrhage, the others from unknown causes; 1 male infant had lived to the age of 6 months, cause of death being unknown; 2 daughters were living at the ages of 3 and 14 years, perfectly healthy from birth. The mother has 2 sisters who have each had 2 male children who died shortly after birth; each of the two sisters has a daughter who is well and strong.

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[1] Clin. Jour., No. 364, 1899; Arch. of Ped., Mar., 1900.

[2] Arch. of Ped., Dec., 1899.

[3] Pediatrics, Nov. 1, 1899.

[4] Indiana Med. Jour., vol. XVIII, No. 5; Arch. of Ped., Mar., 1900.

G. D. Head[1] calls attention to the value of a count of the **leukocytes** as an aid in the diagnosis of diseases in children; 15 illustrative cases are given, and special emphasis is laid on the aid a count of the leukocytes gives in suspected typhoid fever; to differentiate this from appendicitis, osteomyelitis, enterocolitis, or septicemia, the count would be of almost diagnostic value, while if the case is not typhoid fever, a Widal test, being negative, would still leave the diagnosis obscure.

A. Baginsky[2] gives the notes of a case of medullary or **myelogenous leukemia** in a boy 9 years old who had been under observation for a few months. There had been attacks of measles, whooping-cough, and pneumonia, the present illness having begun 2 months before admission. The spleen was enormously enlarged, extending down to Poupart's ligament on the left. The blood examination showed specific gravity, 1046; hemoglobin, 30%; red cells, 2,000,000; leukocytes, 986,000, a proportion of 1 : 2.2. Stained preparations showed poikilocytes and nucleated red cells, and a differential count of the leukocytes gave large mononuclears (myelocytes) 79.1%, polynuclears 12.3%, eosinophiles 5%. The general condition of the boy varied greatly from day to day; sometimes he was comfortable, sometimes almost dying from dyspnea. After a week of fever, the origin of which could not be ascertained, the boy was taken home with a normal temperature. Estimation of total nitrogen and uric acid in the urine showed a decided increase in the elimination of uric acid, the relation being 1 : 30 instead of 1 : 50, probably being directly dependent on the increase in the leukocytes. Arsenic and iron were administered without any apparent effect.

W. N. Bradley[3] reports a case of acute **lymphemia** or acute lymphatic leukemia in a boy 8 years old; there was enlargement of all the superficial lymph-glands and of the liver and spleen; the hemoglobin was 18%, the red cells numbered 1,850,000, the white cells 85,000, a proportion of 22 : 1. The anemia progressed, dyspnea was severe, purpuric spots and hemorrhages appeared, and strength failed rapidly, death occurring after an illness of 8 weeks.

F. Theodor[4] takes exception to most of the cases reported as **progressive pernicious anemia** in children, accepting only 7 as purely cryptogenic and so beyond criticism; to these he adds 1 case in a boy 11 years old; a colored plate illustrates the blood changes and their significance is discussed at length.

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[1] Pediatrics, vol. IX, No. 3.

[2] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.

[3] N. Y. Med. Jour., vol. LXX, No. 26; Arch. of Ped., April, 1900.

[4] Arch. f. Kinderh., Bd. XXVIII, Hefte 5 u. 6.

## DISEASES OF THE URINARY SYSTEM.

L. Concetti [1] reports a case of **sarcoma of the bladder** in a girl 11 months old, and collects from literature 41 cases of tumors of the bladder, tabulating them and analyzing the statistics, which show that even the benign tumors must be treated surgically to give any hope of recovery.

H. Rehn [2] reports 6 cases of **malignant tumors of the kidney,** in 4 girls and 2 boys, 3 being between 1 and 2 years old, one being 5 and the oldest 11 years old. Heredity seemed to play no part, and the tumors had been noticed for periods ranging from several months to, in the oldest, 5 years. The right kidney was affected in every case, hematuria was seen in but one, albuminuria was not observed in any; there were edema of the right leg and dilation of the superficial abdominal veins. Metastasis to the lungs was detected in 2 cases during life. Laparotomy was done in one, metastasis occurring in 2 months. The pathologic diagnosis in 3 cases was sarcoma; in 2 carcinoma, autopsy being refused in one.

E. E. Graham [3] reports the case of an infant dying an hour after birth, having been cyanotic all the time, both **kidneys** being found extensively **cystic.**

F. Bierhoff,[4] writing on **enuresis and "irritable bladder"** in children, is of the opinion that the essential or ultimate condition causing enuresis is hyperesthesia of the deep urethra or sphincter from hyperemia or inflammation, the long list of causes usually given being predisposing or remote; the condition is never a bad habit, and punishment has no place in the treatment; the author recommends hot sitz-baths, the appropriate treatment of accessory causes, restriction of fluids in the evening, and elevation of the hips during sleep; local applications, if possible, are desirable.

H. Huber [5] gives a suggestive paper on the subject of **incontinence** of urine in children and a very useful classification of the causes.

T. J. Elterich [6] reports 2 cases of idiopathic **hematuria,** paroxysmal, and without discoverable cause, either anatomically or in the history; anemia was present, but otherwise there were no symptoms save the blood in the urine. Gull has called such cases "renal epistaxis."

R. W. Raudnitz [7] reports a case of **"minimal albuminuria"** (the persistence for some time of small amounts of albumin in the urine—0.05%) in a girl with gouty antecedents and gouty symptoms; the urine voided in the night and on arising in the morning would be free from albumin, while that passed at noon would have albumin, and in greater amount than the evening urine. The author thinks that the pathologic condition was the gouty diathesis, and that the albuminuria was caused by the meeting of this with certain physiologic conditions, such as erect posture, bodily work, being awake involving mental activity, damp weather, perhaps the taking of food, and that factor on which the course of the body-temperature depends. In the case reported the onset of menstruation caused the albuminuria to cease.

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[1] Arch. de Méd. des Enfants, Mar., 1900.

[2] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.

[3] Arch. of Ped., Oct., 1899.

[4] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.

[5] Arch. of Ped., Nov., 1899.

[6] Arch. of Ped., Mar., 1900.

[7] Arch. f. Kinderh., Bd. XXVIII, Hefte 3 u. 4.

## DISEASES OF THE RESPIRATORY SYSTEM.

J. C. Gittings and C. F. Judson [1] describe under the term **alveolar catarrh** a condition of prolonged irregular fever with cough and expectoration and signs of consolidation at one or other apex, recovery ensuing in from 6 to 8 weeks. Five cases are reported.

A. N. Schkarin [2] investigated the subject of **purulent pleurisy** in infants, with special reference to the bacteriology, reviewing the literature. The general view that empyemas are metapneumonic was borne out by the study of 16 cases in children ranging from 2 to 8 months of age, pneumococci being present in all but three. In 14 cases of serofibrinous pleurisy the pneumococcus was obtained in pure culture every time, the vitality of the germ being less than that in the purulent form; and to this the author ascribes the difference in the character of the exudate. In 20 cases of miliary tuberculosis there was pleurisy, purulent in 9, and serous or seropurulent in the remainder; in 13 the pneumococcus was obtained in pure culture, being present in 4 others with mixed infection; the tubercle bacilli were found by direct examination in the exudate from 2 cases, and by inoculation in 5. These cases were observed in the St. Petersburg Foundling Hospital in the first quarter of 1899, the deaths from tuberculosis during the same period of the past 4 years being 37, 29, 66, and 169.

J. von Bokay [3] reports in detail an **echinococcus cyst of the pleura** in a boy of 5 years, and refers to his previous communication of 3 cases of echinococcus cyst of the liver in children, all 4 cases being successfully treated by Baccelli's method, which consists in the aspiration of the cyst and injection of bichlorid of mercury; shrinkage of the cyst soon follows. None of von Bokay's cases relapsed.

A. M. Vargas [4] gives a clinical and pathologic report of a case of **diffuse latent pulmonary gangrene** in a child of 3 years: Over the base of the left lung there were signs of a massive consolidation, with flatness and absence of breath-sounds; putrid odor, fetid expectoration, and gastric fermentation were absent; the grave general condition did not agree with that of an ordinary pneumonia; pleural effusion was excluded and there was nothing to suggest tuberculosis. At the autopsy the base of the lung was found to be in an eaten-out condition, the necrotic tissue projecting in regular ridges one above another. The etiology, as suggested by Jacobi, is probably an infarct.

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[1] Pediatrics, vol. IX, No. 2.

[2] Jahrb. f. Kinderh., Bd. LI, Heft 6.

[3] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.

[4] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.

H. Weber[1] reports an epidemic of a **contagious form of pneumonic fever** in children; 7 cases were observed, the incubation period being about 12 days, the pneumonia affecting the lower lobes and ending by crisis in 4 days; the sequels were peripheral neuritis, cardiac weakness, and delirium during the decline.

J. L. Morse[2] reports 3 cases of **pneumonia,** in children of 7, 8, and 3 years, the early symptoms being almost exclusively referred to the abdomen, especially in the region of the appendix.

C. Leroux[3] reports **serous meningitis** consecutive to pneumonia in an infant 4 months old, resulting in chronic hydrocephalus; the child recovered, but was backward mentally.

J. S. Meltzer[4] refers to the frequency with which **otitis media** complicates bronchopneumonia, and also to a number of cases of croupous pneumonia in which the initial symptom was a severe earache.

Fever of the inverted type, lower in the evening than in the morning, makes interesting the case of **grippal bronchopneumonia** reported by Faraggi.[5]

J. P. West[6] adds 6 cases to the 2 previously published by him of **enlarged bronchial nodes** giving rise to troublesome cough, cure being effected by creasote. The author believes that the enlargement of the glands is a manifestation of lymphatism and not tuberculous, the tuberculin reaction being negative in some of the cases.

Under the term **urticaria of the mucous membranes,** especially in connection with asthma, F. A. Packard[7] contributes an interesting report of some cases in which fever, dry rales in the chest with distressing cough, and dyspnea would alternate with the appearance of urticarial wheals; and when the latter were out, the chest symptoms would cease. The literature is reviewed. [We may be allowed at this point to refer to a case reported by C. G. Kerley[8] of an angioneurotic edema affecting the tongue and lips, there being a few urticarial wheals on the skin.]

## CONSTITUTIONAL DISEASES.

**Hypothyroidism and Infantilism.**—Hertoghe[9] discusses the action of thyreoidin on infantilism and arrested growth. The normal hypertrophy of the thyroid gland at puberty antedates the development of the sexual organs; the thyroid secretion is increased because of this hypertrophy, and the surplus is used for the growth of the genital apparatus, of which it is only a corollary. The initial cause of infantilism is dysthyroid in nature and complete myxedema is the extreme degree of thyroid degeneration. Dysthyroidism, according to its degree, produces infantilism in children, the stages being: simple obesity, rachitis, chondrofetal dystrophy, infantilism. The fact that dysthyroidism and infantilism have the same etiology is proved by the coexistence of the

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[1] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.

[2] Ann. of Gyn. and Ped., Nov., 1899.

[3] Arch. de Méd. des Enfants, April, 1900.

[4] Phila. Med. Jour., Aug. 5, 1899.

[5] Arch. de Méd. des Enfants, Mar., 1900.

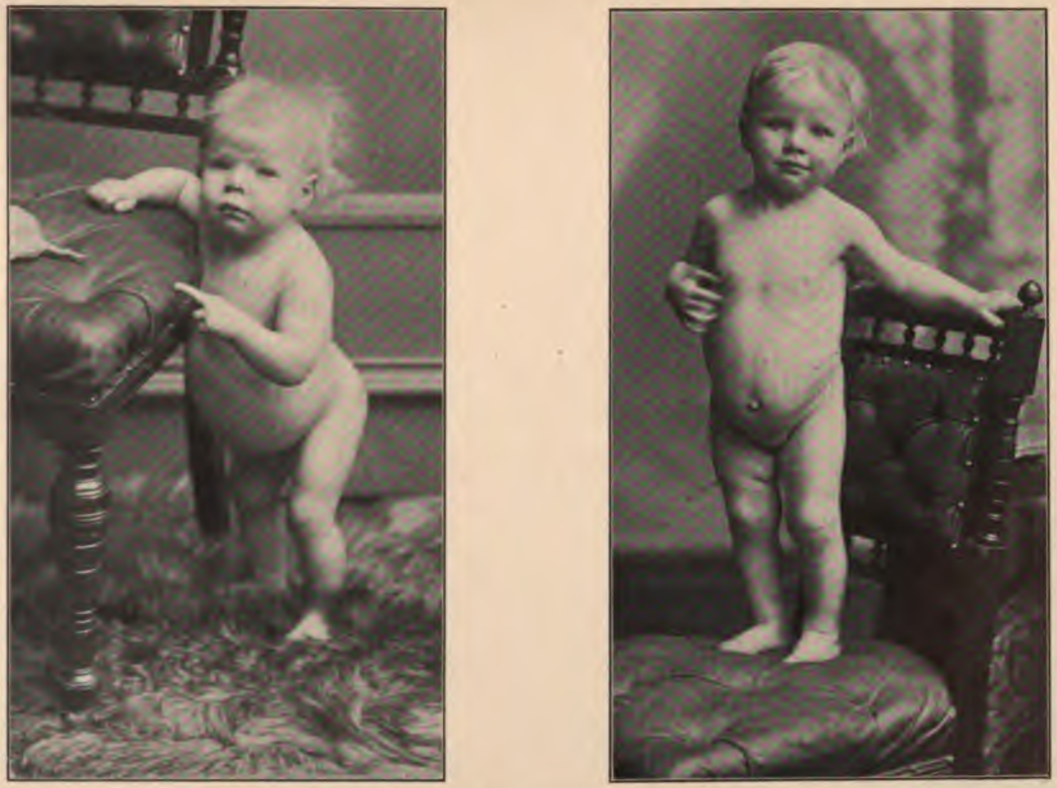
[6] Arch. of Ped., Sept., 1899.

[7] Arch. of Ped., Oct., 1899.

[8] Ibid.

[9] Gaz. des Mal. inf., vol. II, No. 7; Arch. of Ped., June, 1900.

PLATE 2.



Illustrating Nicholson's article on thyroid treatment in a cretin (Arch. of Ped., June, 1900).

different types of infantilism in the same family, by thyroid lesions in the parents (Basedow's disease, thyroid asthma), and by the therapeutic effect of the ingestion of thyreoidin. The same author treats of the subject at length in a monograph which is reviewed extensively by Stoeltzner.[1]

St. Philippe and Guyot [2] report some successful results with **thyreoidin** in backward children. As opposed to Dobrowsky's reports, they saw a striking change in a backward 3-year-old child, who was apathetic and motionless all the time; following treatment, he became lively, spoke, took interest in things, and grew physically and mentally. During an attack of whooping-cough, when the thyreoidin was withheld, development came to a standstill, without there being a relapse; but rapid improvement followed a return to the treatment. Other cases are also mentioned in which marked improvement occurred. In idiots and cases of myxedema there was improvement in the obesity. Loss of weight and emaciation were seen only at the start of treatment, a gain taking place after 2 or 3 weeks, the weight then increasing until it surpassed the original, whether the treatment was continued or not. The preparations of the thyroid gland are energetic stimulants to the system, and while they are specifics in the myxedematous conditions, they are of great effect in infantilism, in rachitis, and in the cachexias of manifold origin seen in backward children. On the latter point emphasis is laid. The doses employed were, at the start, 0.06 gm. (1 grain), increasing in a week to a tablet of 0.2 gm. (3 grains), daily. After 2 or 3 weeks there would be a pause for from 8 to 14 days, and then a repetition of the treatment.

**Cretinism.**—H. O. Nicholson [3] reports the case of a child cretin in which the effects of **thyroid treatment** upon the bodily and mental condition were remarkably rapid and complete. At the age of 2 years and 8 months the child was in the condition of well-marked cretinism. The initial dose of thyroid powder was 2½ grains, once daily; but after 3 days, on account of diarrhea, this was reduced to 1¼ grains for several weeks, when the original dose was given. After 4 months of treatment a photograph, with which the article is illustrated, showed "a bright, happy, pretty child, to all appearances normal, both physically and mentally." (Plate 2.) A few months later death occurred in a malignant attack of measles, but an autopsy could not be obtained. In the etiology the author lays stress on the fact that the child seemed normal for the first 4 months of life, at the end of which it contracted whooping-cough lasting 4 months, and then it was seen to be abnormal. He attributes the origin of the cretinism to the attack of pertussis, and while this is not to be denied at all, further investigations are greatly to be desired in this almost unexplored field.

Under the term **infantile myxedema** A. Muggia [4] reports a case in a boy 7½ years old, with great improvement.

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[1] Jahrb. f. Kinderh., Bd. LI, Heft 6.

[2] Ann. de méd. et de chir. inf., 1898, Nos. 17 and 18; Arch. f. Kinderh., Bd. XXVIII, Hefte 3 and 4.

[3] Arch. of Ped., June, 1900.

[4] Il Morgagni, 1899; Arch. de Méd. des Enfants, Feb., 1900.

Davel,[1] in reporting a similar case in an infant 6 months old, uses the term **athyroidemia,** which, the reviewer says, "is more just and expressive than myxedema, for it localizes the disease and expresses an idea of its pathogenicity."

**Accessory Thyroid Gland.**—Cases are reported by W. Meyjes[2] and by Aschoff.[3] Meyjes' patient was a young woman who had a tumor on the right half of the base of the tongue, which was thought to be an accessory thyroid because no thyroid could be felt in the normal position. Aschoff found at an autopsy on a 6-months-old infant who had died suddenly, having had during life some evidences of myxedema, the site of the thyroid gland occupied by some small gland-like bodies, while at the root of the tongue there was a body which on microscopic examination was seen to be composed of epithelial spaces containing colloid material, and which probably had had a vicarious thyroid-function.

**Rachitis.**—J. L. Morse[4] examined 400 infants seen consecutively in dispensary for signs of **rickets,** and found 80% affected. He considers that a rosary is not a normal phenomenon, but is a constant symptom of rickets—the earliest, and sometimes the only, sign to develop; the next most common symptom is delayed dentition; the other symptoms do not appear, as a rule, before the tenth month. The only apparent common cause was unhygienic surroundings, and not diet or race.

A. Monti[5] contributes a philosophic article on the etiology and pathogenesis of rachitis, showing first by a number of citations that the general opinion of the etiology is harmonious in holding unsuitable nourishment to be the fundamental cause. All kinds of unsuitable nourishment which feed the organism to a certain extent, but which lead to a peculiar form of dyspepsia in which there is in the gastric juice a lessening in the amount of hydrochloric acid and a formation of lactic acid in excess of the normal amount, are, if maintained for some time, calculated to cause rachitis. Breast-fed children may thus become rachitic if the milk is too watery, or too poor in fat, or too low in specific gravity, or too rich in sugar or fat; there need not be any specific changes in the milk, such as Zander found—an increase in the potash salts and phosphoric acid as compared with the sodium salts and chlorin. Bottle-fed children are more frequently rachitic, no matter what method of feeding is pursued, especially if the milk is sterilized. The disturbance of digestion with the decrease in hydrochloric acid and the increase in lactic acid leads to the continual absorption of small amounts of lactic acid and a lessened absorption of lime-salts; the lactic acid has an irritant action on the growing bone tissue, and the resulting increase has not a sufficient amount of lime for calcification.

W. P. Shukowsky[6] noticed that the relative frequency of rachitis diminished steadily in going south from St. Petersburg, where it is present in 95% of all dispensary cases, to 50% in Bessarabia, and he therefore was led to investigate the influence of the relative **humidity** on the appearance of the disease, arriving at the conclusion that rachitis is not dependent on the dampness at all.

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[1] Arch. de Méd. des Enfants, Feb., 1900.
[2] Brit. Med. Jour., Oct. 14, 1899; Jahrb. f. Kinderh., Bd. LI, Heft 5.
[3] Deut. med. Woch., 1899, p. 263; Jahrb. f. Kinderh., Bd. LI, Heft 5.
[4] Boston M. and S. Jour., vol. CXL, No. 7; Arch. of Ped., Jan., 1900.
[5] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.
[6] Arch. f. Kinderh., Bd. XXVIII, Hefte 3 u. 4.

P. N. Sasuchin,[1] as a result of studies on **the spleen in rachitis,** states that it is almost always enlarged except when general atrophy exists; macroscopically, the organ has a thickened capsule and increased consistency, is anemic, and on section shows a reduction in the number of Malpighian corpuscles, with the trabeculæ evident; microscopically, the general picture was that of a chronic interstitial splenitis, some of the islands of connective tissue (the increased trabeculæ) containing small aggregations of lymph-cells; in addition, the Malpighian bodies were smaller than normal, the arteries showed a periarteritis with narrowing of the lumen, and in the center of the follicles there were degenerated epithelioid cells. The author thinks that these changes necessarily interfere with the blood-making function of the spleen, and that they are characteristic of rachitis in cases like his, from which syphilis has been absolutely excluded.

W. Stoeltzner and W. Lissauer [2] failed to find any beneficial effect on the course of rachitis in 6 cases treated with thymus extract for 3 months. Stoeltzner,[3] however, treated 71 rachitic children with adrenal extract, and found it to be of decided value; the symptoms influenced to the greatest degree were the sweating, craniotabes, delay in the appearance of the teeth and in the learning to sit, stand, and walk, the tenderness on handling, the restlessness, and the abnormal vasomotor excitability of the skin. The characteristic odor of the urine observed in rachitis—that of trimethylamin, changing, some time after being voided, to strongly ammoniacal—was very promptly influenced. The abnormal softness of the thorax and the rachitic kyphosis were often bettered. Naturally, from the nature of things little influence was seen on the size of the fontanel, the rosary, the deformity of the thorax, and the epiphyseal enlargements, although some cases showed improvement. In general, the improvement was most rapid in the first week, then slowing down, ceasing when the treatment was interrupted and starting again with the resumption, complications not interfering with the improvement in the rickets. In 3 cases dying of complications, histologic examination showed a cure of the rachitic changes. Harmful effects due to the treatment were entirely absent. Burroughs, Welcome & Co.'s tabloids of compressed gland substance were used, the beginning dose being one-third of a tabloid, amounting to 8 cgm. of gland substance. In discussing the rôle of lime in the pathology of rachitis the author [4] states that a diet poor in lime can not be considered the cause of the disease, and that increasing the amount of lime ingested by a rachitic child merely leads to an increase in the elimination of lime; therefore, neither insufficient absorption nor exaggerated elimination of lime can be held responsible, but that the pathology of rachitis is to be found in the cells which form new bone.

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[1] Jahrb. f. Kinderh., Bd. LI, Heft 3.

[2] Jahrb. f. Kinderh., Bd. LI, 1899.

[3] Jahrb. f. Kinderh., Bd. LI, 1899.

[4] Jahrb. f. Kinderh., Bd. L, 1899.

**Scurvy.**—J. H. Fruitnight[1] adds an interesting note to the reports of cases of scurvy. The patient, a boy of 9 years, had been for years on a diet of ham, bacon, bread, and coffee, never eating fruit; the left knee and wrist and right ankle were tender and swollen, there was a petechial eruption on the body and near the swollen joints, and the gums were spongy, bleeding on pressure; severe pain in the left inguinal region almost caused collapse, necessitating the use of opium. An injection of hot soapsuds and oil brought away a piece of pseudomembrane, 5 by 2 inches, mucoid in character and dark red in color, evidently the result of an ecchymosis of the mucous membrane, the case being therefore scorbutic membranous colitis. Recovery was prompt with correction of diet.

F. M. Crandall[2] details the notes of a case in an infant 6 weeks old, who had been fed from birth at the mother's breast; examination of the **milk,** which was very abundant in quantity, gave 1.8% fat with a **specific gravity** of 1.027, the proteid therefore being very low.

F. Huber[3] refers to the difficulty of diagnosticating mild cases of **scurvy** and to the value of the therapeutic diet test.

L. Guinon[4] describes a case of acute painful rachitis, with slight scorbutic lesions of the gums (concealed Barlow's disease), in an infant 18 months old, fed on "maternized milk." The main symptoms were emaciation, anemia, tendency to lie immobile; pain, apparently constant, which could not be located; edema of the legs, hemorrhages in the gums, disarticulation of the sternum and costal cartilages from the ribs, and laryngospasm; the symptoms of classical scurvy which were absent were subperiosteal hematoma, orbital hematoma, and cutaneous ecchymoses. Recovery followed change of diet and the use of fresh fruit-juice. [We regret the author's desire to return to the use of the term "acute rachitis"; while both are fundamentally disturbances of nutrition, rachitis and scurvy are distinct diseases, although they may coexist; the former being a chronic, the latter an acute disease, when scorbutic symptoms are present the diagnosis should be scurvy with or without rickets, as the case may be, and not acute rachitis.]

A. Bassler[5] gives the macroscopic postmortem findings in a case of scurvy in a child 1 year old.

**Rheumatoid Arthritis.**—R. T. Taylor and S. H. McKim[6] emphasize the fact that rheumatoid arthritis is not allied to rheumatic or gouty troubles in either symptomatology or treatment. Their classification of these joint affections is as follows: (1) Rheumatoid arthritis; (2) chronic fibrous rheumatism, a sequel of subacute attacks of articular rheumatism; (3) arthritis deformans, the general progressive form of Charcot; (4) osteitis deformans of Paget; (5) Heberden's nodosities; (6) partial or monoarticular arthritis deformans. The different types are described and a case is reported of rheumatoid arthritis in a girl 8 years old; photographs and skiagraphs are given.

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[1] Arch. of Ped., Dec., 1899.
[2] Arch. of Ped., Nov., 1899.
[3] Arch. of Ped., Dec., 1899.
[4] Rev. mens. des Mal. de l'Enfance, Nov., 1899.
[5] Phila. Med. Jour., July 15, 1899.
[6] Arch. of Ped., Nov., 1899.

A. Johannessen [1] reports 3 cases of chronic rheumatic arthritis and arthritis deformans in children.

**Diabetes mellitus** in childhood is discussed by S. Bogoras,[2] who reviews the literature very thoroughly, adding to the 130 cases collected by Wegeli, 34 cases from recent literature and 15 unpublished cases. After an analysis of the subject, he calls attention to the importance of heredity in the causation, to the characteristic course, to the almost certain fatal termination, to the constantly negative findings at autopsy, and to the absence of complications.

## DISEASES OF THE NERVOUS SYSTEM.

A. Liebmann [3] urges that the prognosis in **mentally deficient** children should not be given as hopeless without thorough systematic examination, which will often reveal some point upon which successful treatment can be based; case-histories illustrate his points.

M. Thiemich [4] details the examination and the points on which the diagnosis of imbecility in early childhood should rest.

H. Schuschny [5] discusses **brain-fag** in young school-children, the conditions which favor its development, and the measures to be adopted to prevent it.

A. Johannessen [6] discusses the **obscure etiology of acute anterior poliomyelitis,** and refers extensively to the literature; he tabulates observations on 23 cases, and illustrates the article with photographs of 3 patients and skiagraphs of patients and of healthy children of equal ages showing the hypoplasia of the bones; he recommends waiting for a short time after the acute symptoms subside before starting treatment.

Soltmann [7] reports a case the symptoms of which during life were those of pachymeningitis cervicalis hypertrophica, the patient being a girl of 12 years. Postmortem, the membranes were only slightly adherent and not at all thickened, and the cord was found to be converted almost entirely into a **gliosarcoma** with cavity formation and hemorrhages. Colored plates illustrate the article.

G. N. Acker [8] reports a true case of **porencephaly** in a colored boy 4 years old. There had never been control of the right arm and leg, which were wasted. Autopsy showed a large cyst on the left cerebral hemisphere in the region of the Sylvian fissure. The pathologic report by D. Lamb showed that the cyst was in the region supplied by the middle cerebral artery, evidently dating back to early fetal life; alcoholism and perhaps syphilis in the father were the only points bearing on the etiology.

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[1] Zeit. f. klin. Med., 1899; Arch. de Méd. des Enfants, June, 1900.
[2] Arch. f. Kinderh., Bd. XXVII, Hefte 3 u. 4.
[3] Arch. f. Kinderh., Bd. XXVIII, Hefte 1 u. 2.
[4] Deut. med. Woch., No. 2, 1900.
[5] Arch. f. Kinderh., Bd. XXVIII, Hefte 5 u. 6.
[6] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.
[7] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.
[8] Arch. of Ped., Oct., 1899.

R. Cestan [1] refers to the form of **congenital paralysis** described by Little, consisting in the following of premature birth or birth in hard labor by motor troubles, mainly spastic rather than paralytic, and affecting the legs more than the arms, recovery ensuing with the growth of the child, the intellect rarely being disturbed. Little called this the spinal type, as opposed to the cerebral form of infantile paralysis, in which the mental faculties are disturbed; and the name "Little's disease" was given to the former condition. Cestan quotes Marie, Brissaud, and Van Gehuchten as dualists, believing in the existence of the disease as separate from the cerebral palsies in its clinical appearances and morbid anatomy, while Freud, Raymond, and Massalongo are unicists, looking on the condition as merely a variety of the spastic-paralytic infantile palsies. After a consideration of the subject, the author ranks himself with the latter group, and, recognizing that there exists the clinical type, suggests the term "Little's syndrome."

O. Soltmann [2] reports a case of a girl 11 years old, presenting the symptom-complex of **Landry's paralysis,** including partial involvement of the bulb, the symptoms lessening and recovery following mercurial inunctions in 4 interrupted series. The author discusses the diagnosis, and has found references to but 4 other cases in childhood.

M. Thiemich [3] discusses **tetany** and tetanoid conditions in early childhood, and reports 27 cases.

J. Thomson [4] gives as the etiology of **head-shaking with nystagmus,** or spasmus nutans, rachitic infants of the poorer classes, between the ages of 4 and 12 months, living in poorly lighted houses, thus causing eye-strain at a time when coordination is being established, the disease developing in the vast majority of cases in the dark months of December and January.

I. A. Abt [5] reports 2 cases of head-nodding, or **spasmus nutans,** ascribing rickets as the most common cause; the literature is reviewed.

Ausch [6] reports 2 cases of spasmus nutans in children, the history of one bearing out Raudnitz' theory with reference to the darkness of the dwellings in which such children are found to live; in the other case, however, the nystagmus and head-nodding seemed to depend on the fact that the child always lay in one part of the room and constantly held its eyes in one direction in looking toward the light.

G. Hahn, [7] writing on the symptomatology of **cerebellar disease** in infancy, reports the case of a boy 4 months old, of tuberculous antecedents, presenting retraction of the head, which was constantly moved from side to side; the diagnosis of tuberculous tumor of the cerebellum was confirmed at the autopsy.

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[1] Rev. mens. des Mal. de l'Enfance, Oct., 1899.

[2] Jahrb. f. Kinderh., Bd. LI, Heft 1.

[3] Jahrb. f. Kinderh., Bd. LI, Heft 2.

[4] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.

[5] Jour. Am. Med. Assoc., vol. XXXIV, No. 5.

[6] Arch. f. Kinderh., Bd. XXVIII, Hefte 3 u. 4.

[7] Arch. f. Kinderh., Bd. XXVIII, Hefte 3 u. 4.

G. Arnheim[1] reports a glioma of the cerebellum in a 3-year-old boy, causing headache, vomiting, dizziness, choked disc, slight ataxia, convergent strabismus, paralysis of the external rectus, nystagmus, paraplegia, paralysis of the bladder, and finally disturbance of respiration. The literature is reviewed and the diagnosis discussed. Sarcoma of the cerebellum in a girl 4 years old is reported by S. S. Adams.[2]

J. Halle and G. Ulmann[3] report 2 cases of **thrombosis of the cerebral sinuses,** streptococci being obtained from the clots. The first, in a boy of 18 months, was consecutive to a rhinobronchitis and otitis; the other was in a boy 3 years old, who had recovered from pneumonia and double otitis, only to have a relapse of each, during which thrombosis occurred; the symptoms were those of cerebral involvement in pneumonia, and were not characteristic of thrombosis, there being neither edema of the face or eyelids, nor swelling nor pain in the mastoid region, nor dilation of the superficial veins.

L. D'Astros[4] reports a unique case of **tuberculosis** in a boy 13 years old; the tracheobronchial glands and the lungs were first involved, and then there developed a meningitis causing Jacksonian epilepsy, followed by thrombosis of the basilar vein with softening of the cerebral peduncle, causing crossed paralysis, oculomotor on the left and hemiplegic on the right.

C. Comba[5] has investigated the amount of **nitrogen in the cerebrospinal fluid** of children with different diseases, the material collected being as yet not enough to draw decided conclusions from, although 2 cases tend to show that in uremia the total N is decidedly increased, while the albumin remains about normal; it is possible that if this is confirmed it will be a valuable contribution to the differential diagnosis of uremia from many cerebral conditions. The author[6] has also studied the nature of the reducing substance which is present in **normal cerebrospinal fluid** to the extent of 4 cgm. per 100. In grave pneumonic processes it is increased a little, but in meningitis due either to Weichselbaum's meningococcus or to Fraenkel's pneumococcus it is always absent. In tuberculous meningitis it is diminished at the start and disappears toward the end; this diminution and disappearance are probably due to the glycolytic action of the nucleoproteids of the leukocytes, and not to that of the bacteria present. As the proportion of sugar in the fluid is less than that in the blood, the cerebrospinal fluid is therefore a secretion and not a transudation.

F. A. Packard[7] points out that **Kernig's sign** (the inability to extend the leg on the thigh in the sitting posture) in the diagnosis of meningitis did not hold in 3 infants who were proved by autopsy to have had meningitis; the autopsy in one patient who had shown the sign disclosed no anatomic change in the meninges. The author thinks that while the sign may be of value in recognizing meningitis in adults and older children, it is not reliable in infants.

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[1] Arch. f. Kinderh., Bd. XXVII.

[2] Arch. of Ped., Sept., 1899.

[3] Arch. de Méd. des Enfants, Jan., 1900.

[4] Arch. de Méd. des Enfants, Feb., 1900.

[5] Arch. f. Kinderh., Bd. XXVIII, Hefte 5 u. 6.

[6] Clinica Medica, 1899; Arch. de Méd. des Enfants, May, 1900.

[7] Arch. of Ped., April, 1900.

J. B. Herrick [1] found the sign present 17 times in 19 cases, which included 9 of cerebrospinal meningitis, 7 of tuberculous meningitis, 2 of pneumococcus meningitis, and 1 of acute syphilitic meningitis. In 25 healthy persons the sign was absent, and in 100 sick persons, meningitis being excluded, it was found twice, once in a woman who had suffered from gonorrheal arthritis and had been confined to bed a long time, and in another patient, the autopsy showing a subdural hemorrhage without inflammation.

J. Grósz [2] reports a case of **acquired hydrocephalus** in a boy 10 months old, following what was probably meningitis at the age of 6 weeks. There was a convergent downward strabismus, with immobile pupils and amaurosis, the circumference of the head being increased at least 3 cm. Direct aspiration of the lateral ventricle was performed and 40 cc. of fluid were removed, compression being exerted by a bandage; improvement was immediate, the axes of the eyes becoming parallel, the pupils reacting, and vision apparently returning. After 3 days the bandage was removed, and on the next day there was a relapse to the former condition, so puncture was repeated, 70 cc. of fluid being withdrawn; collapse followed this, but the child reacted, and recovery then set in, with normal growth.

J. L. Steven [3] analyzes 112 cases of **chorea,** 87 being seen in the out-patient department and 25 in the wards, the two groups being studied separately. Of the out-patients, 75 % were females, and 43 of the 86 cases occurred between the ages of 6 and 10 years; 2 or more attacks were recorded in 31 %; the duration of the attacks could be fixed definitely in only 36 cases, half being under 2 months. With regard to the causation, definite replies were made in 30, a majority (26) being emotional in origin; note was made of the family history in 56 cases, and in almost 50 % a family tendency to rheumatism, chorea, or some other form of nervous disease could be very distinctly made out; in 26 out of 81 cases there was history of antecedent rheumatism; some evidence of abnormality of the heart was present in 32 out of 82 cases; the urine was practically normal, albumin being found only 4 times; the movements were general in 39 cases, right-sided in 25, and left-sided in 21, and they were severe in 23, moderately so in 35, and slight in 24; of the cases, 31 made only one visit. Of the 25 cases in the wards, 20 were females, 20 were between 5 and 15 years of age, 56 % had experienced previous attacks, and in 18 the duration was over 2 months; fright seemed to cause about one-third of the cases; 11 of the cases had a family history of rheumatism, 1 a family history of chorea, while for 11 the history was quite negative; antecedent rheumatism had existed in 11 and was definitely absent in 8; the heart was abnormal in 15 and unaffected in the 10 remaining; the urine was normal in all but one case, and extended observation of the specific gravity gave an average of 1016; the movements were general in 16 cases, right-sided in 5, and left-sided in 4, while they were severe in 15 and slight in 10; of the 25 patients, 20 were discharged after observation of 2 months or less, 3 remaining for 3 months, and 2 for 4 months.

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[1] Am. Jour. Med. Sci., July, 1899.

[2] Arch. f. Kinderh., Bd. XXVII, Hefte 3 u. 4.

[3] Arch. of Ped., Mar., 1900.

M. A. Starr[1] analyzes the histories of 1400 cases of **chorea** seen in the Vanderbilt Clinic. The proportion of females affected compared with males was almost exactly 2 to 1; the disease is more common in the poorer classes and is accompanied by an apparent anemia. Heredity and infectious diseases seemed to bear no definite relation to the disease, in etiology, the most constant element being the malnutrition. Fright immediately before the onset was noted in 285 cases, and 290 had a distinct history of true rheumatism. Organic heart murmurs were present in 175 cases, functional in 123, and none were heard in 871. Between the ages of 7 and 14 years 919 cases occurred; of 1129 cases, 707 came on between March and August, because of the lack of outdoor exercise during the winter; for this reason also recurrences, which were present in one-fourth of the cases, are most common in the spring. The choreic movements were general in 951 cases, unilateral in 449, the right side being affected slightly more than the left. Mental irritability was noted in 827 and speech was affected in 556. Arsenic, pushed to the physiologic limit and then reduced slightly, is the best drug in the treatment, and antipyrin is second; very little effect was produced by exalgin, phenacetin, bromid, chloral, and paraldehyd. Better than any medicine is a change of air.

C. Gerhardt[2] analyzes 55 cases of **chorea,** and although the number is much smaller than those forming the basis of Starr's paper, the conclusions are in many points strikingly similar, especially as to etiology, the association with acute articular rheumatism (both series giving a lower proportion than is usually stated), the presence of organic heart murmurs, and the proportion of relapses; Gerhardt's cases gave a much higher percentage of heart murmurs than Starr's.

F. J. Smith,[3] as a result of the hypodermic injection of large doses of **arsenic** in chorea, does not think that it has any more specific action than good food and rest in bed have.

G. F. Still[4] reports 3 cases of **day-terrors** (pavor diurnus) in boys of 6 and 3 years and a girl of 4½ years. Suddenly, without any apparent cause, the child begins to scream and look terrified, sometimes imagining that some one is coming after him; there is no loss of consciousness and the duration of the attack varies from a few seconds to about a quarter of an hour. The condition is allied to epilepsy in the sense that it is a paroxysmal neurosis. There was a family history of rheumatism in all 3 cases. Like the night-terrors, the attacks are excited by irritation of the gastro-intestinal tract, especially that form known as mucous disease.

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[1] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.
[2] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.
[3] Clin. Jour., No. 364, 1899; Arch. of Ped., Mar., 1900.
[4] Lancet, No. 3988, 1900; Arch. of Ped., May, 1900.

## MISCELLANEOUS DISEASES.

G. Hahn [1] reports a fatal case of **saturnine encephalopathy** in an infant 13 months old, caused by a 4-months' use of Hebra's ointment for eczema. When the child was seen in the toxic state, convulsions were frequent, and lumbar puncture was done to relieve the intracerebral pressure; improvement followed, but the child died on the following day. In 4.8 gm. of dried brain-substance 0.0013 gm. of lead was obtained. The author reviews the subject of lead-poisoning in children, tabulating the reported cases, giving the age, etiology, symptoms, and termination.

A. Johannessen [2] reports from Christiania the surprising number of 140 children **poisoned by lye** from 1893 to 1898 inclusive. The different features of the poisonings and the symptoms are set forth in tables and the law is quoted which governs the dispensing of strong alkalies.

S. J. Meltzer [3] selected, as appropriate to the volume, the subject of the **toxicology of potassium chlorate from an experimental standpoint,** in view of the fact that Jacobi was the earliest to call attention to the dangers of the drug. The status of previous knowledge was that dogs poisoned with the salt die with methemoglobin in the blood, but that rabbits killed by the same kind of salt have no recognizable change in the blood and there is rarely a nephritis. The author, as a result of intracerebral injections, found that chlorate of potash is a strong poison for nerve-cells, which are first intensely excited and then paralyzed by it.

A. M. Vargas [4] detailed the history of a child 2 years old in a state of general **infantile atrophy,** the weight being 11 pounds, the appearance being like that of a mummy; recovery was rapid on a milk diet, with the important therapeutic measure of the subcutaneous injection of artificial serum twice daily, 100 cc. being given at each injection. Striking photographs, taken before and after treatment, accompany the article.

J. P. West [5] reports a case of **peliosis rheumatica** occurring in a girl 2 years old, there being coincidently evidence of lymphatism.

M. Gil y Casares [6] reports a case of **unilateral hypertrophy of the face** in a boy 9 years old, the increase affecting the right maxillary bones and the right half of the tongue; sensation for touch, pain, temperature, taste, smell, sight, and hearing were defective on the right, but the vasomotor and secretory nerves were unaffected.

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[1] Arch. of Kinderh., Bd. XXVIII, Hefte 3 u. 4.

[2] Jahrb. f. Kinderh., Bd. LI, Heft 2.

[3] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.

[4] Jacobi's Festschrift; Phila. Med. Jour., May 26, 1900.

[5] Arch. of Ped., Dec., 1899.

[6] Arch. de Méd. des Enfants, Feb., 1900.

# PATHOLOGY AND BACTERIOLOGY.

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## TUBERCULOSIS.

**The Cultivation of the Tubercle Bacillus.**—Hesse [1] recommends a new culture-medium for the tubercle bacillus, consisting of 5 gm. of Heyden's Nährstoff, 5 gm. of salt, 30 gm. of glycerin, and 10 gm. of agar, in 1000 cc. of water alkalinized with 5 cc. normal soda solution. On this medium tubercle bacilli can be grown directly from sputum, and colonies can be seen as early as 5 or 6 hours after incubation in an oven.

Fraenkel [2] has reviewed the subject of the cultivation of the tubercle bacillus historically, and has given the results of his own investigations on the cultivation of the bacillus from sputum. He employs Heyden's Nährstoff, as suggested by Hesse. With this and the addition of 2% agar, he obtained a very prompt and luxuriant growth of the tubercle bacillus from nearly every specimen of tuberculous sputum of more than 50 cases examined. If no growth was obtained, it was usually because the sputum had been received in a disinfecting solution. To prevent drying, the Petri dishes are surrounded by a strip of rubber-dam. The sputum must be spread in a thin layer and finely divided, so that it appears like a delicate veil. The growth is visible as early as from 6 to 8 hours afterward, and reaches its height in 7 or 8 days, when the colonies appear as small, grayish-white points. Even better than Heyden's Nährstoff is glycerin serum. This is true for cultures of tubercle bacilli; for purposes of sputum, however, the Heyden Nährstoff agar is superior, and next to that comes an agar made with plasmon. The purification of the sputum by means of heat, for the removal of extraneous bacteria, was not successful; but if the sputum is preserved in glycerin, these latter bacteria are gradually destroyed, without injurious effect upon the tubercle bacilli. [If the method of Hesse proves to be suitable for the rapid cultivation of the tubercle bacillus from sputum, it will become a valuable adjunct to the microscopic examination of sputum as a diagnostic agent, and will also render the cultivation of the bacillus for experimental and serotherapeutic purposes a much simpler matter, particularly when glycerin serum is employed.]

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[1] Zeit. f. Hyg., XXXI, p. 502, 1899.

[2] Hyg. Rundschau, vol. X, No. 13, 1900.

**The Growth of the Tubercle Bacillus in Acid Brain Medium.**—Ficker [1] prepares a culture-medium with extract of brain. The brain, in as fresh a state as possible, is finely divided in a chopping machine, and then slowly raised to boiling-point, in an equal quantity by weight of distilled water. After boiling for 15 minutes the mass is strained through a cloth, and the fluid sterilized for 2 hours by steam. It may be used liquid, mixed with serum in equal proportions, and 3 % glycerin added; or mixed with equal parts of a 2.5 % agar solution in distilled water, to which afterward 3 % of glycerin is added. Brains from different animals, including the human brain, were tried, and no marked difference was found. For the preparation of the serum culture-medium the serum of horses was found to be best.

**The Influence of Oxygen under Pressure on Liquid Cultures of the Tubercle Bacillus.**—F. Arloing [2] finds that oxygen under pressure of from 1½ to 2½ atmospheres has an inhibitory effect upon the growth of the tubercle bacillus, the duration of the exposure having more influence than the degree of compression. Under the influence of the oxygen the virulence of the bacteria may disappear, so that they are no longer capable of infecting rabbits.

**The Resistance of the Tubercle Bacilli in the Sputum to Drying and Decomposition, and Their Modification Regarding Tingibility.**—Lucibelli [3] concludes as follows: (1) The bacilli contained in sputum and dried on glass have a comparatively slight resistance to diffused light (less than 18 days), but a rather strong resistance when kept in the dark (60 to 80 days); (2) bacilli kept in decomposing but fluid sputum in diffused light have a high degree of resistance, and are virulent even after 4 months; (3) the attenuation begins much earlier in sputum kept in closed glass tubes than in that kept in tubes plugged with cotton; (4) the attenuated bacillus when inoculated from one guinea-pig into another does not gain in virulence; (5) when there are only a few bacilli in the sputum, they preserve their tingibility even after prolonged putrefaction, but with gradual attenuation, the decrease in tingibility occurring more readily in closed tubes than in open ones; (6) if the sputum contains very few bacilli, these lose their tingibility as soon as decomposition occurs, or preserve it for only a few days.

**The Local Distribution of Tubercle in Various Species of Animals.**—Woods Hutchinson [4] details the experience gained in the postmortem room of the London Zoological Gardens in about 80 cases of tuberculosis in animals and birds. In all the mammals the lung is the point of severest attack and its lesions the usual cause of death. The bones and joints are very rarely attacked. In the Bovidæ (cattle, antelopes, sheep, gazelles) the disease has a preference for the serous membranes, especially the pericardium and pleura, constituting the well-known pearl disease. In birds the distribution of the lesions is peculiar. Instead of the lungs being the most common site, they are the rarest among the great viscera. The chief site is the liver, next the spleen, and third the wall of the intestinal and mesenteric nodules. The cause of death in tuberculous birds appears most commonly to be diarrhea. Regarding the localization in the lung in mammals, Hutchinson is of the opinion that the infection is not, as a rule, aerial, but alimentary. No matter how the bacteria are introduced, they find their most favorable site in the lungs, because of all great vital organs they are the most recent, in point of formation, in the animal kingdom. As to the curious immunity of the bird's lung to tuberculosis, it can only be said that it is in keeping with its slow susceptibility to diseases in general. [While the lung may evolutionally be an organ of recent formation, we can not see why that predisposes it to disease. In early childhood, for example, the intestinal tract is perhaps more often diseased than any other portion of the body ; and yet the digestive tract is philogenetically one of the earliest systems of the animal kingdom. The vulnerability of the lung, which is probably less than that of the arterial system, is the result, it seems to us, of anatomic and environmental conditions ; if the lungs were ever so old in the scale of life, they would be just as liable to disease as they now are.]

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[1] Centralbl. f. Bakt., Parasit. u. Infektionskrankh., May 4, 1900.

[2] Compt. rend. de la Soc. de Biol., Mar. 30, 1900.

[3] Gaz. degli Osped. e Clin., No. 142, 1899; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[4] Brit. Med. Jour., Nov. 11, 1899.

**The Importance of the Tonsils of Young Children as Portals of Entry for Tuberculous Infection.**—In a thesis which received the prize at the Berlin University, Friedmann [1] describes the results of his investigations on the tonsils of children under 5 years of age, made in 91 autopsies and in 54 living children on whom tonsillotomy had been performed. In the latter, tubercle bacilli were found only once in the tonsils. Among the 91 cases at autopsy tuberculosis was demonstrated, either by characteristic histologic findings or by the discovery of the tubercle bacillus microscopically, in 22 instances. In only one of these did the tonsils seem to be the only part of the body affected, justifying, according to the author, the belief that it was a case of feeding tuberculosis. As, however, in this same case the bronchial glands were enlarged, the conclusion is not entirely without flaw. Unfortunately, the bronchial glands were not examined microscopically. In 3 cases the tissue of the tonsils was free from tubercles and tubercle bacilli, although smear preparations had shown tubercle bacilli on the surface. If animal inoculations had been made from the tonsils in these 3 cases, they would, of course, have given positive results, although the tonsils themselves were not tuberculous. [This leads the author to reject animal experiments as of but little value in determining whether the tonsils are portals of entry in tuberculosis ; but it is hardly likely that any competent experimenter would proceed to inoculate tonsillar tissue without having cleansed the outer surface from adherent bacteria. If that is done, and the deeper portions of tonsils are used, animal experiments are certainly the most trustworthy for the settlement of the questions involved.] Friedmann ascribes tonsillar tuberculosis to two causes : a primary infection through the food and a secondary infection through sputum containing tubercle bacilli. He believes that in childhood tonsillar tuberculosis is most often the result of feeding with tubercular food. [The possibility of this can not be denied, and no doubt it is possible to produce in animals a tonsillar tuberculosis by feeding; but neither the author's studies nor others on the same subject have unequivocally demonstrated the occurrence of feeding tuberculosis of the tonsils in man.]

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[1] Beiträge zur pathol. Anat. u. allg. Path., XXVIII, p. 66, 1900.

**Mixed Infection in Pulmonary Tuberculosis.**—Sata,[1] of Osaka, Japan, working in Ziegler's laboratory, has undertaken a study of the subject of mixed infection and its significance in pulmonary tuberculosis. The study is divided into 6 parts, as follows: (1) A macroscopic examination of the diseased lungs; (2) a microscopic examination of smear preparations from the fresh material; (3) the making of cultures from various parts of the diseased lung; (4) the determination of the bacterial species isolated; (5) a histologic and bacteriologic examination of sections from the hardened lung; (6) animal experiments with tubercle bacilli and other organisms obtained from the lungs. A total of 21 cases of tuberculosis in the human subject was examined. Of these, there were 12 of mixed infection; 6 chiefly with the streptococcus, and 4 chiefly with diplococci. A great variety of bacteria were found, in addition to the well-known streptococcus, staphylococcus, and diplococcus of pneumonia. The pseudodiphtheria bacillus was found in 5 instances, and a bacillus closely resembling it, which the author designates Bacillus pseudodiphthericus pulmonalis. This latter differed from the pseudodiphtheria bacillus in forming a reddish-brown pigment on potato and a faint yellowish-brown pigment on other mediums, and in its very rapid growth. To determine the influence of mixed infection upon animals, rabbits and guinea-pigs were inoculated with tubercle bacilli, intratracheally and intravenously, and subsequently with streptococci and staphylococci obtained from human phthisical lungs. While there was generally a more rapid process, ending in death, the mixed infection did not always influence the course of the tuberculous disease unfavorably. The author's general conclusions are as follows: The bacteria which in the course of pulmonary tuberculosis settle in the contents of cavities, and later in the walls of the cavities, may, through their toxins, exert an influence upon their surroundings or upon the entire body; they may bring about a destruction of the wall of the cavity and lead, either by themselves or in conjunction with the tubercle bacillus, to a pneumonia, in the course of which toxemia and bacteremia may occur; they may also produce, through aspiration into healthy parts, bronchopneumonia, which goes on either to recovery or to a destruction of lung tissue, although it may lead to death through toxemia and bacteremia. Mixed infection presents itself usually in the guise of a pneumonia, which is the result of the co-action of the tubercle bacillus and other bacteria, and assumes either a lobular or a lobar character. Microscopic examination reveals streptococci, staphylococci, and pneumococci, either with or without tubercle bacilli. Mixed infections play a prominent rôle in phthisical processes, and influence not only the clinical course, but likewise the pathologic process in the lung tissue. A positive demonstration of mixed infection is possible only by histologic examination in conjunction with cultural methods. The examination of the sputum and the cultivation of bacteria from the lungs of cadavers are in themselves not sufficient to demonstrate mixed infection. Mixed infection usually occurs after the disintegration of the tuberculous tissue has begun, and may not occur until some time afterward. The contents of cavities remain free from foreign bacteria only when the cavities are closed; usually they become infected as soon as there is a communication with the external air, but this does not as yet constitute mixed infection. That occurs only when the bacteria penetrate into the wall and bring about pneumonic processes in the neighborhood, or when they are carried by aspiration into distant parts and set up a mixed pneumonia. What is called pulmonary phthisis is generally a pure tuberculosis only in the beginning. Pure forms of tuberculosis of the lungs which progress but slowly and are after a time arrested, and eventually are discovered at autopsy, can neither in their clinical course nor in their anatomic structure be designated as phthisis; although in cases of advanced tuberculous processes with localized pneumonic exudation about the foci, fibroid areas occur in which mixed infection is not demonstrable. Whether in them mixed infection never occurred, or whether the secondary infection has disappeared and the tuberculosis only remains, can not be decided anatomically. The majority of advanced cases of phthisis are mixed infections, and a large part of the phthisical changes is the result of such secondary infection. Pure local tuberculosis of the lung runs an afebrile or a slightly febrile course, while mixed infection brings about high fever; so that the presence of such fever permits the conclusion that mixed infection exists. Pathologically the difference between pure tuberculous phthisis and that complicated with secondary infection is not qualitative, but only quantitative, the inflammatory process in the latter being much more marked. The aggravation of the disease through mixed infection can be demonstrated in animals. The chief bacteria concerned in mixed infection are Streptococcus pyogenes, Staphylococcus pyogenes aureus, Diplococcus pneumoniæ, the pneumobacillus and its varieties, and Bacillus pseudodiphthericus pulmonalis. Not all of the bacteria found in the sputum or by culture at autopsy are concerned in the destruction of the lung. The lesions produced by secondary infection may undergo resolution, so that finally only the tuberculosis remains. It is probable that the secondary infection does not always intensify the phthisical process; it may act as a hindrance to the multiplication and spread of the tubercle bacilli and may bring about changes that lead to cure. The tuberculous foci do not usually enlarge from the center, but through coalescence of contiguous foci. The single focus has a tendency to heal; the danger of spreading lies in the formation of new foci through transportation of bacilli. The wall of a cavity is not often tuberculous, but consists of a well-formed granulation tissue. This feature is probably connected with a secondary infection, in which the organisms previously named displace the tubercle bacilli.

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[1] Beiträge zur pathol. Anat. u. allg. Path., III, Supplementheft, 1899.

**Experimental Tuberculosis in a Goat.**—Ravenel and White [1] succeeded in producing tuberculosis in a goat by inoculating the animal through the chest-wall into the substance of the lung with a pure culture of bovine tubercle bacilli. [While the conditions under which the disease was produced were purely artificial and practically impossible in nature, they show that the goat is not tuberculosis-proof. Occasionally the disease occurs under natural conditions also. These facts should be remembered in employing goat's milk for infants' food.]

**The Action of Dead Tubercle Bacilli.**—Kelber's [2] experiments seem to show that dead tubercle bacilli have no specific action, and are incapable of producing a characteristic tubercle. Intravenous injection of such bacilli produces inflammatory changes only in the lung. Leukocytes and a few epithelioid cells accumulate, but rarely giant cells, and there is no tendency to caseation.

**The Passage of the Tubercle Poison from the Parents to the Offspring.**—Maffuci [3] concludes from his investigations: (1) That the tubercle poison may pass to the child with the sperm, with the ovum, and through the placenta; (2) that the offspring of two tuberculous parents is most likely to be tuberculous; (3) that the embryonic intoxication manifests itself in poor development, abortion, miscarriage, death, or cachexia in extra-uterine life; (4) that the embryonic tissues resist the development of the tubercle bacillus and are even able to destroy it; (5) that chicks developed from infected eggs possess greater resistance to the virus than chicks or adult chickens developed from healthy eggs; (6) that children of tuberculous parents are not more susceptible to tuberculosis than children of healthy parents, and that the greater frequency of tuberculosis in the descendants of tuberculous parents is due to infection in the family, or, perhaps, to the possible presence of embryonic germs, and is not the result of an inherited predisposition; (7) that congenital tuberculosis may heal in a favorable environment; (8) that even embryonic tuberculosis may improve or be cured in the new-born under good hygienic conditions. The tuberculosis question can be solved only by the strictest hygiene; the children must be removed from the focus of infection in the family and placed in healthful surroundings.

**The Passage of the Tubercle Bacillus into the Milk of a Tuberculous Woman.**—H. Roger and M. Garnier [4] relate the case of a woman, aged 34 years, who was being treated for pharyngeal tuberculosis, and in whom an area of consolidation was observed at the summit of the left lung. The patient was pregnant and was soon delivered, and from that time the symptoms became aggravated and she died 17 days after her labor. The autopsy showed tuberculosis of the liver, kidneys, and thyroid body. Two days after its birth the child presented icterus, then green diarrhea, and finally a hard edema of the feet, hands, legs, and thighs. The child had at first been fed on the bottle, but at the request of the mother it was fed at the breast for 2 days and its condition was made distinctly worse. It died 6 weeks after birth and the autopsy revealed tuberculosis of the liver, the spleen, the kidneys, and the mesenteric lymph-nodes. The bacilli were found in these lesions. Four days after the birth of the child 2 guinea-pigs were inoculated with milk taken aseptically from the breast of the mother. One of the animals received 4 cc. of the milk under the skin, while the other received 2 cc. into the peritoneal cavity. The first animal died, and at autopsy typical tuberculous lesions of the liver, the spleen, and the lungs were found. The second animal lived and increased in weight, but it presented enlarged inguinal lymph-nodes. It was killed, and at autopsy there was a fibrous peritonitis at the point of inoculation, the loops of intestines were adherent to each other and to the abdominal wall, the large omentum was retracted, and the surface of the liver showed spots that resembled cicatrices. Bacilli were not found in any of the organs. The child was undoubtedly infected from the mother, and the location of the lesions seems to point to the digestive tract as the principal, if not the sole, source of the infection. [This observation emphasizes the importance of tuberculous mothers not nursing their children.]

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[1] Proc. Path. Soc. of Phila, April, 1900.

[2] Arb. aus dem pathol. Institut in Tübingen, 1899, II, Heft 3; Centralbl. f. allg. Path. u. pathol. Anat., Jan. 2, 1900.

[3] XIV. Congress della soc. di chir.; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[4] Compt. rend. de la Soc. de Biol., Mar. 2, 1900.

**Serum Diagnosis of Tuberculosis.**—As the tubercle bacillus is nonmotile and not uniformly distributed in liquid culture-mediums, it lends itself less readily than the typhoid or cholera bacillus to serum diagnosis. It is claimed, however, by Arloing and Courmont[1] that by frequent agitation of glycerin-peptone bouillon cultures a more homogeneous distribution and a certain degree of motility are attained. Many precautions are, however, necessary : the culture must be transplanted monthly, and always in the same amounts, to an identical medium. The bacilli undergo striking modifications—to such an extent, indeed, that they are scarcely recognizable. If left unagitated, they eventually revert to their original form. In the performance of the test an 8- or 12-day-old culture is used, the material for the test being taken from the upper portion of the culture. The authors obtained positive reactions in all cases of tuberculosis except those with virulent and advanced infection.

Bendix [2] tested the **serum reaction** in 38 cases of tuberculosis, and obtained negative results in only 2, both of them being advanced cases of phthisis. The milder cases gave the most pronounced reaction. In 3 healthy persons no reaction was obtained. Hence he considers the serum reaction of value in the early diagnosis of tuberculosis.

Fraenkel [3] was unable to confirm the observations of Arloing and Courmont on the **agglutination of the tubercle bacillus** by serum from cases of tuberculosis. In 8 cases in which tuberculosis was suspected there was no reaction at 1:10 ; at 1:5 and 1:3 there was a moderate reaction in 2 cases. In 7 patients with indubitable tuberculosis, and with tubercle bacilli in the sputum, the reaction was obtained only once at

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[1] Jour. de phys. et de path. gén., II, 1900.

[2] Deut. med. Woch., April 5, 1900.

[3] Hyg. Rundschau, vol. X, No. 13, 1900.

1 : 10 and once at 1 : 5. Twenty persons with other diseases were examined, 9 of whom gave the Widal reaction. Fraenkel, moreover, did not observe the motility of the bacillus in Courmont's cultures. [While a final judgment on the method is not yet possible, the results so far achieved offer very little promise of the agglutinating test as at present practised ever becoming of diagnostic value in tuberculosis.]

**Bacillus of Pseudotuberculosis.**—The bacillus of pseudotuberculosis was isolated by Pfeiffer from rodents affected with a disease closely resembling tuberculosis. Klein [1] discovered the organism in Thames and Lee water, and also in sewage. It was easily cultivated, and morphologically was a short bacillus. Large quantities were introduced into the subcutaneous tissue of guinea-pigs. An ulcer formed at the site of injection and the neighboring lymph-glands became caseous. Feeding experiments were also productive of positive results. The disease could be reproduced in monkeys from pure cultures. By the treatment of animals with dead cultures resistance could be brought about.

## SYPHILIS.

**The Etiology of Syphilis.**—Schüller [2] in a brief communication states that he has found in all syphilitic lesions, even in the hereditary form, a parasite belonging to the protozoa. He was able to cultivate it, although with difficulty, on account of the presence in the majority of syphilitic glands and chancres of contaminating micro-organisms. [A fully illustrated article is promised. Until that appears conclusions are not justified. The universal presence of the protozoon is, however, in itself sufficient to raise a doubt as to the author's discovery.]

Van Niessen [3] has isolated a bacillus from the condylomata and blood of syphilitic patients with which he claims to have produced a disease in monkeys characterized by primary lesion, polyganglionic indolent buboes, constitutional symptoms with lesions of skin and mucous membranes; finally, fatty liver and icterus, periarteritis and ischemic lesions in the central nervous system, etc. The bacillus resembles the pseudodiphtheria and the Lustgarten bacillus. Despite numerous flaws, which the patient author himself recognizes,—such as the brevity of the incubation period in animals and the paucity of the experiments,—he is still of the opinion that he has discovered the cause of syphilis. [Van Niessen's former observations were proved to be fallacious, as he himself finally recognized. While it is to be hoped that he has at last been successful, much stronger evidence is necessary to establish his claim.]

**Changes in the Blood-vessels in Syphilis.**—The majority of writers on syphilitic disease of the blood-vessels have confined their studies to the cerebral arteries, and the view has gained vogue that syphilis affects only the vessels of the brain. In addition to showing that syphilis may attack the arteries of other organs, Abramow's[1] studies also throw light on the nature of the changes that take place in the vessels. The first case reported is that of a youth of 19 years, without a history of syphilis, who died of Bright's disease. At the autopsy the following macroscopic lesions were found: Generalized arteriosclerosis, multiple small subpericardial aneurysms, cirrhotic kidneys (the seat also of parenchymatous nephritis), acute lobar pneumonia, and edema of the brain. Microscopic examination of sections from the heart showed a high degree of thickening of the intima of the blood-vessels, the media and adventitia being for the most part normal. In the liver the vessel-walls were uniformly thickened. The renal vessels presented changes similar to those of the heart. The endothelium was entirely gone. The second case was that of a young student of 25 years who had acquired a chancre 2 years before his entrance into the hospital, and who had received active antisyphilitic treatment. His symptoms were those of interstitial nephritis, with hypertrophy of the heart and arteriosclerosis. There was also a multiple neuritis. The autopsy revealed sclerosis of the coronary arteries, generalized arteriosclerosis, chronic interstitial nephritis, edema of the brain and its membranes and of the lungs; pleural adhesions on the right side. The vessels of the heart presented, on microscopic examination, localized internal thickenings, with scattered involvement of the adventitia. In the coronary artery the thickened intima was on its inner side the seat of hyaline degeneration; there was also mucoid change in the new tissue of the intima. The pulmonary vessels presented nothing abnormal, nor did those of the thyroid gland. In the liver the intima of the blood-vessels was hyperplastic; this was also true of the kidney, of the stomach and intestines, and of the suprarenal glands. The vessels of the brain showed thickening of the intima, but of a trifling degree when compared with the changes in the other organs. Although in the first case there was no history of syphilis, the similarity of the vascular changes to those in the second case, with a distinct syphilitic history, lead the author to look upon these changes also as syphilitic in nature. [The history is so meager that certain other factors can scarcely be ruled out, although alcohol played apparently no part. The possibility of chronic lead-poisoning has to be considered in such cases, as well as heredity, apart from hereditary syphilis.] Careful study of the blood-vessels showed that any one of the 3 coats might be the seat of change—the intima presenting hyperplasia of the endothelium; the adventitia, round-cell infiltration; and the media, granular degeneration. When the intima and adventitia are diseased, they may grow toward each other, penetrating the wall in doing so. As a rule, the intimal changes precede those of the adventitia. Staining of the elastic tissue after Weigert showed it to be for the most part normal; occasionally it was thickened. The formation of a new fenestrated membrane, as claimed by Heubner and by Wendeler, was not observed by Abramow—only a splitting of the old. In the second case the cardiac and renal vessels showed in the thickened intima mucoid, granular, and caseous areas. Caseous areas were seen in the adventitia of the peripheral arteries. This is interesting, as it was claimed by Heubner that regressive changes did not occur in syphilitic arteritis. Abramow looks upon the caseous areas of the adventitia as degenerated gummas. There is, in his opinion, nothing specific in the vascular changes in syphilis, except in the gummatous form. Syphilis may produce a diffuse hyperplasia of the intima, but the syphilitic nature of this can not, in the absence of gummas, be recognized by the microscope.

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[1] Path. Soc. of London, Nov. 7, 1899; Brit. Med. Jour., Nov. 11, 1899.

[2] Centralbl. f. Bakt., Parasit. u. Infektionskrankh., April 20, 1900.

[3] Wien. med. Woch., Nos. 11–14, 1899; Centralbl. f. Bakt., Parasit. u. Infektionskrankh., April 20, 1900.

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[1] Ziegler's Beiträge zur pathol. Anat. u. zur allg. Path., Bd. XXVI, Heft 2.

**Intestinal Syphilis and Syphilitic Endophlebitis.**—Klebs maintained that syphilitic disease of the small intestine begins in the form of small gummatous foci in the submucosa, which gradually enlarge and permeate the various coats of the intestine. If they reach the surface, ulcerations follow; or they may be absorbed, with the formation of stellate scars. Johnson and Wallis, on the basis of 19 cases,—among them 7 personal observations,—expressed the opinion that while Klebs' view is correct in some cases, in others the process begins as a diffuse infiltration of the superficial layers of the intestinal wall, commencing in the mucosa and submucosa. Forssman[1] reports an instance of syphilitic disease of the intestine in which the disease, instead of being gummatous, partook of the nature of a chronic fibroid process. The patient, a woman of 23, had suffered much from diarrhea, the nature of which was not understood. She died of marasmus, and at the autopsy 14 strictures were found in the small intestine. The mucous membrane showed a few ulcers, the floors of which were formed by a smooth, radiating, fibrous tissue. Tubercles were not visible. The mesenteric glands were enlarged. In the colon there were a number of follicular ulcerations, which were probably of a dysenteric nature, and not due to the syphilis. The mesentery corresponding to the region of the strictures was thickened. One gumma was found at the edge of one of the strictures. The mucous membrane above the first stricture was the seat of a diffuse infiltration with small round cells. There were also small, vascular, round-cell foci, which were evidently beginning gummas. On the margins and the floors of the strictures vascularized cell-accumulations, containing also giant cells, were found. There was no necrosis, and tubercle bacilli were absent. The vessels of the intestinal wall in general were not altered, but those in the affected parts showed thickening of the arteries and veins, particularly of the adventitia; and in the arteries, to some extent, of the media and the intima. In the mesentery the veins showed most striking lesions, characterized by papillomatous thickening of the intima, producing in some instances complete obliteration. Evidently the process began about the vessels of the submucosa of the intestine, and led to the formation of connective tissue, ending in strictures. The author believes that some of the strictures were produced without a previous formation of gummas, but in others it was impossible to say whether gummatous formations had or had not preceded them.

[1] Beiträge zur pathol. Anat. u. zur allg. Path., Bd. XXVII, Heft 2, 1900.

**Congenital Syphilis of the Gastro-intestinal Tract.**—Oberndorfer [1] concludes from a study of the visceral forms of congenital syphilis with especial reference to the gastro-intestinal tract: (1) Syphilitic disease of the gastro-intestinal tract is very rare. In exceptional instances the disease occurs in the form of gummas. (2) The point of departure of the gummatous new formation is almost exclusively the submucosa, and here usually the blood-vessels. (3) The follicular apparatus, as a rule, bears no relationship to the diseased portions of the intestine, but it does not follow from this that follicles or plaques are not to be detected in the neighborhood of diseased areas. (4) The extent of the diseased areas increases in the lower portions of the intestine, in the colon and rectum, so that these may present large surfaces the seat of ulceration. (5) Only those diseases of the intestinal tract may be looked upon as luetic that present thickening of the intestinal wall and alterations of the blood-vessels, and in which bacteriologic investigations yield negative results. (6) It is only by a microscopic examination of the tissues that syphilitic disease of the intestinal tract may be detected.

**Syphilis of the Liver.**—Oberndorfer [2] has carefully studied the liver from a little girl who died of hereditary syphilis when about 16 months old. The microscope showed changes characteristic of a marked hypertrophic cirrhosis, with much destruction of the liver tissue and its replacement by a cellular connective tissue. The arteries were in part normal and in part almost obliterated by proliferation of the intima, and the adventitia was also in places permeated by round cells. The veins were even more extensively diseased than the arteries, a fact chiefly shown in penetration of their walls by newly forming connective tissue. The biliary channels were in places proliferated and surrounded by new connective tissue. In the portions replaced by connective tissue numerous miliary nodules with central caseation and surrounded by nuclear fragments and round cells were seen. There was a distinct line of demarcation between normal and diseased tissue. Over the diseased portions the capsule of the liver was thickened. The elastic tissue was greatly increased. Macroscopically, the lesions were found localized on the lower portion of the anterior surface, extending down toward the base near the lobus Spigelii. This distribution is peculiar, since, as a rule, syphilis causes a diffuse process in the liver. Another peculiarity was the absence of lesions at the transverse fissure, where ordinarily they are most marked. Another interesting feature of the case was the incorporation of suprarenal tissue in the liver.

**The Pathologic Anatomy of Leprosy.**—Sokolowsky [3] details the results of a necropsy performed upon a subject dead of leprosy and of the examination of a number of skin-nodules removed from patients during life for diagnostic purposes. The organs examined were the skin, peripheral nerves, spleen, liver, kidney, ovary and tube, and lung. With regard to the skin, the epidermis (with the exception of a single nodule) was entirely normal, and revealed the lepra bacillus in no preparation. The typical microscopic picture—and one, it is stated, that serves for diagnosis—consists of a narrow (0.02 mm. to 0.07 mm. wide) layer of tissue, poor in nuclei and free of bacilli, situated in the upper part of the cutis and surmounting the layer of cellular infiltration wherein the bacilli are to be found in large numbers. In the upper part of the cutis the cellular infiltration, which consists of a variety of cells, is arranged in islets or cord-like bands that reveal an intimate association with the hair follicles and the overfilled blood-vessels. Below, the islets of infiltrate become confluent to form large leprous areas in the subcutis. While some bacilli were found free in the tissues, for the most part they were intracellular—irregularly distributed in the protoplasm or collected into clumps. A large number of bacilli were found in the endothelium of the vessels, and sometimes also in free leukocytes and desquamated endothelial cells within the vessel lumen, but no bacilli were extracellular in the vessels. None were to be found in the sweat-glands, sebaceous glands, or hair follicles. In one case they were found in the erector pili muscle. The alterations of the epithelium of one nodule consisted in the formation of vacuoles. This was attributed to trophic alterations, and was interpreted as the commencement of leprous pemphigus. The alterations in the nerves were those of an interstitial neuritis. The slightest changes consisted in a scarcely perceptible thickening of the perineurium; prominent endothelial cells containing bacilli in the lymph-spaces; and club-shaped or spindle-shaped cells containing bacilli elsewhere in the connective tissue of the endoneurium. When further advanced, the changes consisted in thickening of the perineurium, proliferation of the connective tissue of the endoneurium, and a greater number of endothelial cells and cells containing bacilli infiltrating the connective tissue. In the most advanced stage, which revealed itself by spindle-form swellings of the nerves evident to the unaided eye, the changes consisted in excessive thickening of the perineurium, marked proliferation of the endoneurium, and a diminution in the number of nerve-fibers. The bacilli were present in greater number and distributed throughout the section—in the perineurium, the endoneurium, and the fibrous sheaths, but chiefly in the proliferated tissues. They could not be detected in the nerve-fibers themselves. In the spleen the bacillary invasion was very rich and followed almost exclusively the blood-vessels and the trabeculæ. Vacuoles described by other authors could not be detected. The bacilli were both extracellular and intracellular. In the liver and kidney the bacillary invasion was marked, and the bacilli were also intracellular as well as extracellular.

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[1] Virchow's Archiv, vol. CLIX, p. 179.

[2] Centralbl. allg. Path. u. pathol. Anat., Mar. 15, 1900.

[3] Virchow's Archiv, vol. CLIX, p. 521, 1900.

**Chronic Glanders in Man.**—Baracz [1] reports a case of chronic glanders in man which is characterized especially by its long duration

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[1] Virchow's Archiv, vol. CLIX, p. 491, 1900.

—15 years. Infection is thought to have occurred through the extraction of a tooth by a shoemaker, but it is admitted that shortly after the extraction the patient may have come in contact with the specific infectious agent. Of this, however, there was no evidence. The period of incubation was very short—but several days. The disease was characterized by the occurrence of nodules on the face, chin, and neck, pustules on the nose, and ulceration of the nasal mucous membrane. Interesting features of the case were the recurrence of the manifestations in spring and autumn, and the fact that the patient is said to have been free from the disease for a period of 5 years following the removal of a number of softened cervical glands. At the end of the five years the symptoms became much aggravated and acute. Granulations in the nose grew excessive, and the resultant embarrassment of breathing was relieved for but a short time by their removal—such was the rapidity of their growth. The cervical or submaxillary lymph-glands were markedly implicated in the disease process. Late in the course of the affection there developed nodules and fistulous abscesses on the trunk and upper and lower extremities. The fever was of the continued remittent type. Under increasing cachexia and asthenia the patient ultimately died. The diagnosis in the case was confirmed by microscopic and bacteriologic examination of the pus from the nodules and by the results of animal experimentation. The effect of mallein was not investigated on account of the condition of the patient and because of certain untoward results following its use. It is believed that the value of this preparation is a matter to be determined by future experimentation.

**The Ray Fungi and Their Relation to Certain Bacteria.**—A review of the present state of the perplexing question of the ray fungi and their relation to other bacteria, particularly the tubercle bacillus, is given by Hektoen.[1] The tubercle bacillus and the diphtheria bacillus have both been shown occasionally to assume branching forms, but the occurrence is so rare that, while demonstrating a certain degree of relationship, particularly of the tubercle bacillus, to the ray fungi, it does not as yet afford a basis for ultimate classification. The author, however, agrees with Hueppe that the usual designations, bacilli and bacteria, are not adequate to represent the situation correctly and scientifically.

**Actinomycosis of the Human Lung.**—A case of pulmonary actinomycosis in a woman is reported by Flexner.[2]

**Primary Actinomycosis of the Kidney.**—A case of this rare affection is reported by O. Israel.[3] The patient was an infantry officer 33 years of age, whose symptoms were hematuria and backache, and later anemia. A nephrotomy was done, but without relief. Swelling eventually developed, and incision revealed pus with granules characteristic of actinomycosis. The kidney was removed; the pelvis contained a stone. The upper pole of the kidney was occupied by a grayish, glistening tumor, looking not unlike a carcinoma. Microscopic examination showed fungus-rosets. The stone proved to be nothing more than a mass of calcified fungus in a matrix of pus. The portal of entry of the actinomyces was not ascertained, but it is worthy of note that all the patient's molars were in a state of advanced caries. [Apparently human actinomycosis is on the increase. The existence of the disease should be kept in mind, as in that way an incorrect diagnosis, at the bedside or in the autopsy room, can at times be avoided.]

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[1] N. Y. Med. Jour., May 26, 1900.

[2] Proc. Path. Soc. of Phila., April, 1900.

[3] Verhandl. d. Berl. med. Gesellsch., 1900, xxx, 1, p. 282.

**Leptothrix in Chronic Enteritis and Progressive Pernicious Anemia.**—Damateis,[1] in 1896, found in the feces of a patient who had pernicious anemia and a mild chronic intestinal catarrh a species of leptothrix in large numbers. Recently he has observed a similar case. He found the organism during the diarrheal periods, and not when the intestinal functions were normal. As its presence was associated with intestinal epithelium, which was intertwined with the leptothrix threads, he concludes that leptothrix may damage the bowel epithelium. He believes that it may also obstruct the chyle vessels and prevent the absorption of chyle. The possibility of intoxication by its metabolic products is also admitted. In that way it might be the cause of a metamorphosis of a chronic enteritis into a progressive anemia.

## TYPHOID FEVER.

**A Simple Method for the Diagnosis of Typhoid Fever.**—Piorkowski [2] has found urine mediums of great advantage in the differentiation of the typhoid and the colon bacillus. The medium recommended is prepared as follows: Urine (specific gravity 1020) collected for 2 days, and slightly alkaline from decomposition, is mixed with 0.5% peptone and 3.3% gelatin, boiled for 1 hour on the water-bath, and immediately filtered without further heating. It is then distributed into test-tubes, which are sterilized in the steam sterilizer at 100° C. for 15 minutes. The sterilization is repeated once on the following day for 10 minutes. On this medium the colon cultures, after 20 hours' growth at 22° C., present themselves, under a low power of the microscope, as round, yellowish, finely granular, sharp-edged colonies; the typhoid cultures as radiating filaments. The plates *must* be kept at 22° C., or else the typhoid cultures do not develop typically. In making cultures from typhoid stools, Piorkowski introduces two œses of the feces into one tube; from this, 4 œses into the next one; and from this, from 6 to 8 into the third. In all the cases of typhoid fever examined—there were only 4—he was able to make a diagnosis within 20 hours. In one of them the Widal test was not positive; in another he succeeded in demonstrating the bacilli 3 days after defervescence.

**A New Culture-medium for the Isolation of the Typhoid and the Colon Bacillus.**—Mankowski [3] prepares an infusion of toadstools instead of meat and adds 1.5% agar, 1% peptone, and 0.5% salt.

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[1] R. Accad. di Torino, Mar. 10, 1899; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900

[2] Verhandl. d. Berl. med. Gesellsch., 1900, Bd. XXX, 2, p. 68.

[3] Centralbl. f. Bakt., Parasit. u. Infektionskrankh., Jan. 6, 1900.

Bacillus coli grows rapidly in the form of a silver-white, firm, dry pellicle; the typhoid bacillus grows more slowly, as a transparent, shining, moist streak. The difference between the two is rendered more striking by the development of gas, produced in 24 hours by the colon bacillus. If the medium is colored, as suggested by the author,[1] the two behave in the manner indicated. Both edible and poisonous fungi can be used to prepare the medium.

**The Value of Hankin's Method for the Cultivation of the Typhoid Bacillus.**—Hankin's[2] method is as follows: Fivebouillon tubes are charged with a few drops of the fluid to be investigated. The first remains as a control to the others. One, 2, 3, and 4 drops respectively of Parietti's solution (carbolic acid 5, hydrochloric acid 4, water 100) are added, and all tubes placed in the incubator. After 24 hours the tubes are more or less turbid. One showing a uniform turbidity is selected and is employed for the insemination of another series of 5 tubes. To the first of these as many drops of Parietti's solution are added as the one selected contained. If this was 2 drops, the second series of tubes receive 2, 3, 4, 5, and 6 drops. After another 24 hours a third series may be inoculated; usually this is not necessary, and it suffices to inoculate agar tubes from one showing a uniform turbidity. From the agar tube the colonies resembling those of the typhoid bacillus are transferred to litmus milk. The tubes that after from 1 to 2 days' incubation show a change to red are discarded; the others are further studied, microscopically and by the agglutination-test, to prove their typhoidal nature. Hankin succeeded by his method in isolating the typhoid bacillus from relatively pure and also from dirty water. Hilbert[3] has employed Hankin's method, and comes to the conclusion that it is serviceable in the absence of Bacillus coli, and enables the isolation of the typhoid bacillus from water to be accomplished. If, however, the colon bacillus is present, the method fails.

**Rapid Differentiation of the Cultures of Typhoid and Colon Bacilli.**—Mankowski[4] has found that these organisms alter colored culture-mediums in different ways. The coloring solution is prepared as follows: Solution A is a 1 % solution of caustic potash saturated with acid fuchsin. Solution B is a saturated watery solution of indigo-carmin. To 2 cc. of solution A and 1 cc. of solution B, 22 cc. of distilled water are added. The mixture should be dark blue and feebly alkaline. Enough is added to the culture-medium—*e. g.*, agar—to give it a blue or bluish-violet color. The nutrient medium must be accurately neutral. Under the influence of the typhoid bacillus the medium becomes raspberry-red, while the colon bacillus changes it at first to a bluish-green, and then decolorizes it entirely.

**Changes in the Cardiac Ganglia in Typhoid Fever.**—Strada[5] examined the cardiac ganglia in 10 cases of typhoid fever and found

[1] See note [4].
[2] Centralbl. f. Bakt., Parasit. u. Infektionskrankh., XXVI, p. 554.
[3] Centralbl. f. Bakt., Parasit. u. Infektionskrankh., April 20, 1900.
[4] Centralbl. f. Bakt., Parasit. u. Infektionskrankh., Jan. 6, 1900.
[5] Bull. soc. med. chir. di Pavia, 1899, II; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

congestion of all the blood-vessels, with cloudy swelling of the endothelial cells, and quite frequently endarteritis and endophlebitis. Foci of small-cell infiltration, typhoidal lymphomas, were also seen. In the nerve-cells chromatolysis was frequent. Vacuolization also occurred. The nucleus was at times swollen and hydropic; frequently it was displaced peripherally.

**Bacteriology of the Lobar Pneumonia in Typhoid Fever.**—In 2 cases of lobar pneumonia in typhoid fever Stühlern [1] isolated the typhoid bacillus from the sputum. In one of the cases the bacillus was also obtained during life from the fluid aspirated from the lung. No typhoid bacilli were found in the blood taken from a vein. In both cases the sputum also contained the diplococcus of pneumonia; and the author believes, as most observers do, that the lobar pneumonia of typhoid fever is chiefly due to the pneumococcus, and that the typhoid bacilli present have but a secondary importance. They may have something to do with the tendency of the pneumonia to become markedly hemorrhagic.

**The Distribution of the Typhoid Bacillus in the Human Body.**—Guizzetti [2] has examined 48 cases of typhoid fever at autopsy to determine: (1) The usual portal of entry of the bacilli; (2) the path along which the bacilli spread; (3) the organs that are their point of election; and (4) their accidental sites. The typhoid bacillus was obtained from 43 of the cases. The bacilli are found in those lymph-glands of the mesentery into which the lymph-vessels from the ulcerated portion empty, and also in those which are intimately connected with these lymphatics. They are not present in the lymph-glands of unaffected portions of the intestine. In the glands, the lymph-sinuses and the gland tissue constitute the habitat of the bacilli—not the blood-vessels. The bacilli may pass along the whole lymphatic channel, from the intestines to the receptaculum chyli and thoracic duct, and it is through this channel that they enter the body in general. Examination of other groups of lymph-glands outside of the abdomen seems to show that the first localization of the typhoid bacilli is in all probability the intestine, and that they spread elsewhere through the lymph-paths. The author therefore concludes that typhoid fever is in ordinary cases an infection through the alimentary tract.

**The Presence of Typhoid Fever Bacilli in the Blood of Typhoid Fever Patients.**—In 16 cases of typhoid fever Castellani [3] found 4 (3 of them fatal) in which the typhoid bacillus was found in the blood.

**The Presence of the Bacillus of Typhoid Fever in the Urine of Typhoid Patients.**—Terrili [4] found that the typhoid bacillus might be in the urine without the occurrence of albuminuria, although its presence is frequently accompanied by functional or anatomic changes in the kidney. The bacilli may be found at any period, even in the first week and during convalescence. The quantity eliminated with the urine may be so large that it exceeds that found in the feces.

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[1] Centralbl. f. Bakt., Parasit. u. Infektionskrankh., Bd. XXVII, Mar. 23, 1900.

[2] Assoc. med. chir. di Parma, Dec. 1, 1899; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[3] Accad. med. physica Fiorentino, Jan. 16, 1899; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[4] Tenth Ital. Cong. of Internal Med.; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

**The Prevention of Typhoid Fever by Inoculation with Typhoid Vaccine.**—Marsden [1] has seen a number of cases of typhoid fever in the medical and surgical nursing hospital staff, but since using inoculation with Professor Wright's typhoid vaccine no cases have occurred among the staff. The constitutional symptoms following the inoculation lasted 36 hours, and consisted of headache, general pains, at times nausea, and even vomiting. [The results of typhoid vaccination in the British army in South Africa were very satisfactory, and suggest the advisability of practising the method in all cases where large bodies of troops are collected together in unhygienic localities.]

Wilson's [2] experience with Wright's typhoid serum has been very satisfactory. He found that while it will not render the typhoid bacillus innocuous, it at least modifies favorably the attack of typhoid fever. He claims also to have observed the beneficial influence of the serum on gonorrhea and gleet.

**Contribution to the Study of the Origin of the Typhoid Antibodies.**—Deutsch,[3] after a study of the origin of the antityphoid bodies, concludes: (1) That a single intraperitoneal injection of a typhoid culture into a guinea-pig results in the formation of antibodies. (2) That the antityphoid power appears in the serum at about the fourth or fifth day; it increases until it reaches the maximum at about the eleventh or twelfth day. It then diminishes, but is still present a month after the injection. (3) That the antityphoid power is small in the liver, the kidney, the suprarenal bodies, and the omentum. The value of the antityphoid power of the peritoneal exudate sometimes approaches that of the serum, but never surpasses it. (4) That in from one-fourth to one-fifth of the cases the bone-marrow, and in one-half of the cases the spleen, is more active than the serum. (5) That the lymphoid organs have some relation to the formation of the antibodies, but very often (one-third of the cases) they take no part in the formation of the preventive bodies. It is supposed, then, that these bodies are formed elsewhere; possibly in the blood itself. (6) That the rôle of the said organs is demonstrated by the following facts: (*a*) the development of the antityphoid property in animals deprived of the spleen a long time before immunization is the same as in normal animals; in the majority of these cases the bone-marrow is more efficient than the serum; (*b*) splenectomy performed on animals during the first days of their immunization is followed by a considerable diminution of the antityphoid power; sometimes this effect of late splenectomy is not pronounced; (*c*) the injection of spleens thus extirpated into the peritoneum of other guinea-pigs results in the formation of specific agglutins in the blood of the latter animals, which proves that typhoid substances are fixed in the spleen. (7) That the formative cells have not been discovered; the place of the formation of the antibodies only has been indicated. The lymphatic character of these places (blood, spleen, bone-marrow), on one hand, the great variability of the facts observed, on the other hand, lead to the supposition that migratory cells of leukocytic origin, charged with microbic products, form the preventive antibodies. The second part of the paper treats of the origin of agglutins and their relations to the antibodies, the author drawing the following conclusions: (1) That the intraperitoneal injection of a heated typhoid culture results, in the guinea-pig, in the appearance of an agglutinating power of the serum. (2) That the appearance and the development of this power are subject to the same rules as the development of the antibodies. (3) That the two curves generally progress at a like pace, but they are not superposable. Agglutinating serums in high dilution are always preventive; but there are serums with a weak agglutinating power that nevertheless contain antibodies in large quantity. The parallelism is not absolute, and the identity of the agglutins and the antibodies can not be sustained. The agglutinating power can not be considered as being the basis of the immunizing power; the two are associated in the majority of cases, but not always. (4) That the liver, the kidneys, and the suprarenal bodies of immunized animals contain only traces of agglutins. (5) That the lymphoid organs (spleen, bone-marrow, and lymph-nodes) may contain variable quantities of agglutins without lessening the agglutinating value of the serum. (6) That splenectomy preceding the immunizing injection does not prevent the formation of agglutins; splenectomy done from 3 to 5 days after the injection lessens the formation of agglutins, which will, in the majority of cases, appear in quantity below the normal. The spleen may contain the products of micro-organismal growth that result in the formation of agglutins. The rapidity of the transformation of agglutogenic substances into agglutins can be determined only by comparative examination of the organs and the serum; the agglutins may be secreted, passing into the blood, or it may be that they are formed in the blood itself. (7) That the lungs may be considered as the only organs in the guinea-pig that possess, in the majority of observed instances, an agglutinating value higher than that of the serum. This action of lung extract, not being specific, should be considered as absolutely independent of the action of the specific agglutins of immunized serum. The pulmonary agglutins are easily found in the lungs of normal animals of different species, as well as in the new-born guinea-pig. In contact with cultures they are precipitated, agglutinating different micro-organisms (typhoid bacillus, colon bacillus, plague bacillus, and cholera bacillus) as well as protoplasmic particles of cellular origin. They are not destroyed by drying, but are only slightly resistant to heat. Certain analogies indicate that the normal agglutins are derived from the pulmonary agglutins. (8) That the juice of the fresh lung is the first animal humor known that, while being strongly agglutinating, does not contain preventive bodies.

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[1] Brit. Med. Jour., April 28, 1900.

[2] Brit. Med. Jour., April 28, 1900.

[3] Ann. de l'Inst. Pasteur, Sept., 1899.

**The Value of the Agglutination Test as a Means of Diagnosis of the Typhoid from the Colon Bacillus.**—Horrocks[1] believes that if a powerful antityphoid serum is used, the quick examination by the hanging drop at once brings out marked differences between the true typhoid bacillus and allied coliform organisms. If a weak serum is employed, the specific action is so slow that the hanging drop method quite fails to differentiate between the organisms, and it is necessary to prolong the examination for 24 hours, when distinct differences in the agglutination become apparent.

**Persistence of the Agglutinating Power in Typhoid Serum Preserved in the Liquid State.**—C. Nicolle and A. Halipré[2] have studied the agglutinating power of 3 specimens of typhoid serum which they had kept for 3 years in sealed pipets containing a small quantity of air. The first specimen presented an agglutinating power of 1 to 10 at the end of 15 months and at the end of 3 years. The second specimen presented an agglutinating power of 1 to 50 after the same lapse of time. The third specimen was a mixture of serums from a number of patients, and presented an agglutinating power of 1 to 60 at the end of 15 months and 1 to 40 at the end of 3 years. The specimen was contaminated.

**Experimental Peripheral Neuritis Produced by the Typhoid Toxin.**—H. Vincent[3] produced a rapid general paralysis, simulating the syndrome of Landry, in a rabbit by injecting a culture of the typhoid bacillus associated with a saprophytic bacillus isolated from the human intestine. The rabbit presented pronounced alterations of the spinal cord and of the peripheral nerves of all four limbs.

## YELLOW FEVER.

**Bacillus Icteroides and Its Toxin.**—Lacerda and Ramos[4] claim that they were able to produce with cultures of Sanarelli's bacillus the typical symptoms of yellow fever in rabbits and dogs. The toxin of the bacillus is a protoplasmic poison, producing a fatty degeneration in the heart, liver, and kidney; it also influences the sympathetic nervous system and the muscle-fibers. Serum therapy has so far failed, because the artificial serum is antibacterial, and not antitoxic, and falls short of effect because the symptoms are due to a toxin.

Sanarelli[5] answers Sternberg's criticism, denying that a case has been made out for the assertion of the latter that the hog cholera bacillus and Bacillus icteroides are identical. He insists, supported by the observations of Achinard, Pothier, Wasdin, Geddings, and Woodson in America, and of others elsewhere, that Bacillus icteroides is the specific cause of yellow fever. In his answer to the foregoing, Sternberg[6] quotes the conclusions of Agramonte[1] and those of Reed and Carroll, still unpublished. The former are as follows: (1) Bacillus icteroides has no more to do with the production of yellow fever than the common colon bacilli frequently found in the blood and viscera of yellow fever subjects. (2) When proper methods of cultivation are employed, the bacillus of Sanarelli is usually not obtained in cultures from yellow fever patients. (3) The bacillus of Sanarelli is also found in the tissues of persons dead of other diseases that have nothing to do with yellow fever. (4) The bacillus of Sanarelli is not agglutinated by the serum of yellow fever patients or convalescents. (5) The serum of yellow fever convalescents affords no protection against infection with Bacillus icteroides. Reed and Carroll's conclusions are: (1) Bacillus X of Sternberg belongs to the colon group. (2) Bacillus icteroides of Sanarelli is a member of the hog cholera group. (3) The modes of infection, the duration of the disease, and the lesions in mice, guinea-pigs, and rabbits are the same for Bacillus icteroides as for the hog cholera bacillus. (4) The clinical symptoms and the lesions produced by intravenous inoculations of Bacillus icteroides into dogs are also produced by the hog cholera bacillus. (5) Bacillus icteroides when fed to hogs causes a fatal infection, characterized by diphtheric and ulcerative lesions in the intestinal canal such as are seen in the same animal when infected with the hog cholera bacillus. (6) Hog cholera may be acquired by hogs when the animals are placed in pens infected with Bacillus icteroides or when they are fed with the intestines of infected hogs. (7) Guinea-pigs may be immunized against hog cholera by means of sterilized cultures of Bacillus icteroides, and vice versa. (8) Rabbits may be immunized against hog cholera by means of gradually increasing doses of living cultures of Bacillus icteroides. (9) The serums of animals immunized with Bacillus icteroides and with hog cholera bacillus have a reciprocal agglutinating action. (10) While the blood of yellow fever patients has no agglutinating action on Bacillus icteroides, that of animals infected with hog cholera agglutinates the organism, and this points to a greater affinity of the latter with hog cholera than with yellow fever.

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[1] Brit. Med. Jour., April 8, 1900.
[2] Compt. rend. de la Soc. de Biol., Feb. 2, 1900.
[3] Compt. rend. de la Soc. de Biol., Mar. 16, 1900.
[4] Arch. de Med. expér. et d'Anat. patholog., XI, No. 3, 1899.
[5] Centralbl. f. Bakt., Parasit. u. Infektionskrankh., Feb. 7 and 12, 1900.
[6] Centralbl. f. Bakt., Parasit. u. Infektionskrankh., June 9, 1900.

## THE PLAGUE.

The **plague bacillus** was discovered almost simultaneously by the Japanese, Kitasato, and the Frenchman, Yersin, in 1894. It is a short bacillus with rounded ends, occurring in the tissues singly or in pairs, and in cultures often growing in chains—the so-called streptobacillus form. It does not stain uniformly, the central portions often remaining unstained, on account of which it shows the so-called polar staining so often met with in the diphtheria bacillus. Some doubt exists as to whether or not the organism is motile. Gordon[2] states that the bacilli have flagella and are motile. Their motility has also been asserted by Ibrahim Bey.[3] McFarland[4] does not consider them motile. Most authorities maintain that the organism does not form spores, although Ibrahim Bey states the contrary. It is readily cultivated on all mediums, but on none does it possess any unequivocal characteristics, although its growth in bouillon has certain peculiar features that facilitate its recognition. If it is inoculated into flasks of bouillon and the bouillon is kept absolutely at rest, flakes form on the surface, and from them a growth extends downward in the form of pointed pendants—the so-called stalactites. Slight agitation of the flask causes the stalactites to sink to the bottom as a powdery deposit. The organism does not produce gas or acid. It grows best at the temperature of the body, although it will develop at room-temperature. On dry agar it develops involution forms, which Haffkine considers more or less characteristic.

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[1] Med. News, Feb. 10 and 17, 1900.

[2] Lancet, 1899, I, p. 688.

[3] Méd. moderne, No. 75, 1899; Centralbl. f. Bakt., Parasit. u. Infecktionskrankh., May 4, 1900.

[4] Proc. Path. Soc. of Phila., 1900, vol. III, No. 8.

The plague bacillus is exceedingly virulent, and is probably more dangerous to work with in the laboratory than any other bacterium, on account of the fact that it may penetrate through the slightest wounds and abrasions of the skin, through mucous membranes, through the respiratory tract, and, according to some authorities, even through the unbroken integument. Great care is therefore necessary on the part of laboratory workers and assistants if the tragic experiences of Vienna are to be avoided. There is, however, no valid reason or wisdom in absolutely forbidding, as Austria has done, the scientific study of the bacillus. This is the more true as the outbreaks in Oporto and in Glasgow necessitate active efforts on the part of Europe and America to meet the disease at the earliest moment, and to combat it if it should gain a foothold. The rules to be observed in the construction and management of plague laboratories are clearly and precisely set forth by Markl.[1] The bacillus is pathogenic for rats, mice, guinea-pigs, and rabbits. The first seem to be naturally susceptible, and die in vast numbers immediately before and during an epidemic. Kitasato and Yersin were able to isolate the specific bacillus from rats that had died spontaneously. It is highly probable that these rodents are largely concerned in the spread of the disease, both *in loco* and to distances. The disease can be carried to countries that are not naturally its habitat by various means—through the medium of ships' cargoes, and through human beings suffering from the plague, particularly in a mild form. In the case of the Oporto epidemic, in Portugal, it was apparently introduced through a sick sailor. Rats infected with the bacilli may be carried on shore in cargo, particularly grain, or they may reach the wharves along cables and hawsers. This fact has been recognized and precautions against the landing of rats have been taken. Thus, at Durban the rats were thwarted by the placing of funnels over the hawsers; and although the ship in question came from a plague-infected port, the disease gained no foothold in Natal. According to Mattei,[2] mice are also spontaneously susceptible, and may carry the disease.

In the **diagnosis of plague** the clinical features are important, but their discussion is beyond the province of this section. As a rule, particularly in early cases, the diagnosis must rest on bacteriologic evidence. As the cultural properties of the plague organism are not very characteristic, some other method must be employed, the best being intraperitoneal inoculation of a guinea-pig. Müller and Pöch,[1] following Albrecht and Ghon, recommend the rubbing of the suspected culture into a shaved area of skin of a guinea-pig.

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[1] Centralbl. f. Bakt., Parasit. u. Infecktionskrankh., May 4, 1900.

[2] Bollet. delle Sed. dell' Accad. Gioenia in Catania, LVII–LVIII, 1899; Centralbl. f. Bakt., Parasit. u. Infecktionskrankh., Jan. 15, 1900.

The **serum treatment of plague** was inaugurated by Yersin, who, in 1895, in collaboration with Calmette and Borrell,[2] endeavored to secure an antitoxin by injecting into animals toxins from cultures of plague bacilli. Not being successful, he injected animals intravenously with bacilli previously heated to 58° C., and thus killed. The animals injected became more or less immune. When the inoculation was practised subcutaneously, the immunity developed more slowly, but was more certain. The serum from a horse treated in this way was capable of immunizing guinea-pigs and mice to plague and also of curing the disease. In man, the serum was first used in Canton,[3] and later at Amoy, when 23 cases were treated with serum, all but 2 recovering from the disease. It was found that the injections had to be given early to be beneficial—on the first day about 30 cc. of serum sufficed ; if the patient was seen later, larger quantities were necessary. Wysskowitz and Zabolotny [4] proved the efficacy of the serum in monkeys. Mason [5] used Yersin's serum in a large number of cases in the hospital at Cutch Mandavi, with a mortality of 60 %. In 100 patients not treated with serum the death-rate was 85 %. Out of 721 persons inoculated with the serum for prophylactic purposes, none developed plague—a very striking result. Nazareth [6] treated 47 cases with a serum prepared by Roux after Yersin's method, and had a mortality of 46.8 %. The treatment was practically of no avail in the pneumonic form of the disease. Clemon [7] had very little success with Yersin's serum—of 50 cases treated with it, 40 died ; and of 50 treated without, 40 died. Zabolothy [8] used it in 10 cases, with 6 deaths. Yersin,[9] in the epidemic of Annam, employed the serum in 33 patients, with 19 recoveries—a mortality of 42 %. Of 39 cases not treated with serum, all died. In the Oporto epidemic Calmette and Salimbeni [10] treated 142 patients with serum, with a mortality of 14.78 %. During the same period, out of 72 cases not treated with serum, 46 died—a death-rate of 63.72 %. Calmette and Salimbeni recommend the intravenous injection of 20 cc. of plague-serum in every case of plague as soon after the beginning of the disease as possible, to be followed by two subcutaneous injections of 40 cc. each within the next 24 hours. The immunity conferred by serum injections lasts about a fortnight, so that it is necessary to repeat the injection every 2 weeks.

Besides Yersin, another man has won fame in connection with the struggle against the plague—namely, Haffkine,[1] who does not use the serum of horses inoculated with toxin or dead bacteria, but the dead bacteria themselves. The bacteria are killed by heating the culture for 1 hour to 70° C. The prophylactic fluid, as it is called, is made by cultivating luxuriant crops of the plague bacillus by adding to the nutrient mediums abundant quantities of fat exposed to free aeration. The fat used is clarified butter, known in India as "ghee," and is deposited on the surface of the ordinary culture-mediums in large flasks. Growth occurs in the form of stalactites, as already mentioned. On shaking the flasks, they fall to the bottom, and another group forms. In this way 6 crops may be obtained in about 4 weeks. The sediment produces a local inflammation and a nodule at the seat of the inoculation, and is accompanied by little fever. The fluid, on the other hand, produces considerable rise of temperature with no noticeable local effects. The organisms are killed by exposing them to a temperature of 70° C. for 1 hour. On January 23, 1897, plague broke out in the House of Correction, Byculla, Bombay, when the number of inmates was 345. In 6 days 9 cases occurred, with 5 deaths. On January 30th 6 new cases developed, of which 3 proved fatal. On the same afternoon 154 prisoners volunteered to undergo the preventive inoculation, and each one received 3 cc. of the mixture of sediment and fluid. At the time of the inoculation one of the men had a swollen gland and two others developed buboes a few hours after the inoculation. These patients died. From this time up to February 6th 12 new cases of plague appeared among those of the inmates who were not inoculated, 6 of which were fatal; but among those inoculated only 2 cases appeared, and both patients recovered. The prophylactic appears to act in from 12 to 14 hours. Between January 10 and May 7, 1897, 11,362 individuals from the plague-infected areas were inoculated. Of these subjects 12 died, 3 of whom were sick when they were inoculated, 3 of whom contracted the disease within 12 hours, 2 of whom fell ill within 3 days, and 4 of whom were attacked from 15 to 25 days after the inoculation. Of the subjects, 33 developed plague and recovered. A rough comparison made at the time seemed to show that the noninoculated persons contracted plague 20 times more frequently than those that were inoculated. In February, 1897, medical men in the plague districts in India began to be inoculated with Haffkine's prophylactic. Haffkine's[2] next experiment was made in Umerkadi jail, where the plague broke out late in December, 1897, and by January 1, 1898, 3 prisoners were attacked, all of whom died. All the 401 prisoners were willing to be inoculated, but only one-half were allowed to receive the inoculation, and then the inoculated and the uninoculated were allowed to live and to eat together.

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[1] Spec. Path. u. Therap., Bd. v, Th. 4.

[2] Ann. de l'Inst. Pasteur, 1895, p. 589.

[3] Ann. de l'Inst. Pasteur, 1897, p. 81.

[4] Ann. de l'Inst. Pasteur, 1897, p. 662.

[5] Birmingh. Med. Rev., Sept., 1897.

[6] Brit. Med. Jour., Feb. 11, 1899.

[7] Brit. Med. Jour., Jan. 7, 1899.

[8] Ann. de l'Inst. Pasteur, 1899, p. 833.

[9] Ann. de l'Inst. Pasteur, 1899, p. 251.

[10] Ann. de l'Inst. Pasteur, 1899, p. 865.

After this, prisoners of both the inoculated and the uninoculated groups were discharged daily, and so lost to observation; but of those that remained up to the end of the plague epidemic there were 127 uninoculated and 147 inoculated. In the uninoculated group 10 cases developed, with 6 deaths; 3 cases developed in the inoculated group, which were mild in character and from which the patients recovered. In the Dharwar jail 373 prisoners were inoculated after 5 fatal cases had occurred; after the inoculation only 1 case developed, and the patient recovered. In Undhera, in a free population, on February 12, 1898, the plague had proved fatal to 79 victims. Then one-half the members of each family were inoculated. Subsequently, plague visited 28 families, in which there were 64 uninoculated members and 71 inoculated members. The 64 uninoculated persons had 27 cases of plague, with 26 deaths; while the 71 inoculated persons had 8 cases, with 3 deaths. Later, inoculations were made in Lanowlie, 700 people; the followers of the artillery in Kirkee, 1530; Damaon, 8230; in Hubli, Dharwar, and Gadag, 80,000. From this work, although the results became more inexact the larger the number inoculated, it was found that the mortality in the inoculated was from 77.9% to 90% lower than in the uninoculated. The immunity lasts from 4 to 6 months. In Hubli, Leumann [1] inoculated 24,000 persons twice and 10,000 persons once with Haffkine's prophylactic, between May 11 and September 27, 1898. As a result there was a protection of 85% during the whole epidemic. Clark [2] stated before the special plague commission that of 1407 inoculated persons at Umbala, none contracted the disease. Cawthorn [3] made 38,000 inoculations at Gadag, of which 18,000 were double. She used a dose one-fifth stronger than the prescribed dose in three-fourths of the cases. Of the number that received the stronger dose, 86 persons took plague. Out of the remaining one-fourth of the persons inoculated with the usual dose, 66 took plague. The British Medical Journal's [4] analysis of the evidence given before the plague commission indicates that the witnesses who had tried Yersin's serum all agreed that it was ineffective.

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[1] Brit. Med. Jour., June 12, 1897.

[2] Brit. Med. Jour., July 1, 1898.

Galleotti [5] holds that protective inoculation is the only method of prophylaxis and the only means of combating a spreading epidemic. Instead of Haffkine's method, which he approves in principle, he recommends the inoculation of the nucleoproteid extracted by him and Lustig from the plague bacillus. This nucleoproteid has been proved to be harmless to human beings, and has advantages over Haffkine's prophylactic. The latter's fluid contains, in addition to the active substance, toxic materials, is liable to contamination, and its dose can not be accurately determined.

Terni and Bandi [6] employ the following method of preparing a prophylactic fluid: They inoculate guinea-pigs in the peritoneal cavity, collect the seropurulent exudate that forms, dilute it with physiologic salt solution, sterilize it by repeated heating at from 50° to 52° C., add sodium carbonate and carbolic acid, and use the resulting mixture for injections. For human beings the dose would be from 2 cc. to 2.5 cc. They have seen prompt immunity in animals without the grave reactions following the use of Haffkine's prophylactic.

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[1] Brit. Med. Jour., Sept. 2, 1899.

[2] Brit. Med. Jour., Jan. 28, 1899.

[3] Brit. Med. Jour., Mar. 11, 1899.

[4] Brit. Med. Jour., April 29, 1899.

[5] Lo Sperimentale, 1899, III; Centralbl. u. allg. Path. u. pathol. Anat., July 18, 1900.

[6] Preliminary communication, Messina, 1899; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

**The Transmission of Plague to Hogs, Sheep, and Poultry.**—De Mattei [1] found that hogs, sheep, and birds (pigeons, chickens, ducks, sparrows) were not susceptible to plague. Hogs and sheep may become sick in consequence of the introduction of large quantities of infectious material, but they do not die. Birds are always immune.

**The Resistance of the Bacillus of Plague.**—Bandi and Stagnetta-Balistreri [2] found that the plague bacillus could survive and remain virulent for 15 days in grain deposits. In food products, particularly salted meat and fish, the bacillus dies in a few days. This occurs also in pure and impure water. In the cadavers of rats it remains virulent for at least 2 months. Cargoes on board ships constitute a grave danger, not so much *per se* as through the rats which they attract.

Sata [3] studied 4 different stocks of plague bacilli, and found that, aside from differing in virulence, they were practically the same. They were all decolorized by Gram's method, showed no motility, and had no capsules in cultures, nor in smear preparations from the tissue juices of diseased animals, but showed a halo in tissue sections. They did not curdle milk.

## DIPHTHERIA.

**The Relation of Dextrose and the Production of Toxin in Diphtheria Cultures.**—Theobald Smith [4] from a series of experiments draws the following conclusions: (1) Dextrose is not in itself injurious, but rather favorable to toxin production. When added in quantities not exceeding 0.2% to peptone bouillon freed from fermentable, acid-producing substances (muscle-sugar), it leads to maximum accumulation of toxin by utilizing the available peptone to the best advantage. (2) The different courses taken by different cultures of diphtheria bacilli in ordinary unfermented peptone bouillon containing muscle-sugar, and in peptone bouillon made from fermented infusion to which 0.1% to 0.2% dextrose has been added, are manifested by an increased production of toxin in the latter as well as by a rapid return from an acid to an alkaline reaction. In the former an acid reaction may prevail even under most favorable conditions. (3) These differences may be explained by assuming either that the acid products of the muscle-sugar are different from those of dextrose and nonutilizable, or else that the bouillon contains certain other unknown inhibitory substances removed during fermentation. The use of synthesized mediums, and an analysis of the acid products in fermented bouillon plus dextrose and in unfermented bouillon, would aid in explaining the differences. (4) Among the accessory conditions which favor the toxin production in unfermented bouillon, as pointed out by Park and Williams, are increased quantities of peptone, well-developed surface growth of the diphtheria bacilli, and a low initial acid reaction (phenolphthalein). In fermented bouillon these accessory conditions are also favoring, though of less importance.

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[1] Riv. d'Igiene e Sanita pubbl., No. 18, 1899; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[2] R. Accad. Peloritana di Messina, July 11, 1899; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[3] Arch. f. Hyg., 1900, XXXVIII, 2, p. 105.

[4] Jour. of Exp. Med., May-July, 1899.

**The Constitution of the Diphtheria Poison.**—The researches of Ehrlich have shown that bouillon cultures of the diphtheria bacillus contain, besides the specific toxin, so-called toxoids. These toxoids are bodies resulting from a modification of the toxin; they have not the power of killing animals, but preserve that of fixing the antitoxin. The toxoids do not all have the same avidity for the antitoxin, and they may, therefore, be divided into (1) the prototoxoids, which have a greater avidity for the antitoxin than has the toxin; (2) the syntoxoids, which possess an equal avidity with the toxin; and (3) the epitoxoids, or toxons, which possess less avidity than the toxin. The toxin is also known as the toxophorous substance, and the toxoids are known as the haptophorous substance. Madsen,[1] by experiments upon 4 specimens of diphtheria poison, has proved the existence of these two substances. His experiments have shown that the more or less complete transformation of the toxins into toxoids is accomplished without modification of the degree of affinity for the antitoxin. In two of the specimens the quantity of the poison that was neutralized by an immunizing unit remained unchanged, while the volume of the toxin unit increased in an appreciable manner. The change in the toxin takes place according to certain definite laws. One of the properties of the toxons is to produce late paralyses. The fact that the paralyses are produced by the toxons explains the reason why paralysis is seldom observed in animals that have survived experiments for the determination of the toxicity of a given diphtheria toxin; while they are frequently seen after experiments for the standardizing of serums according to the old method of Ehrlich. The edema produced by the toxons differs from that produced by the toxin in its shorter duration and in the absence of necrosis and of alopecia at the point of inoculation. The toxons, however, do not always present the same properties, and consequently the time of the death of the animal is a more satisfactory indication of the pathogenic property of these bodies than is the production of edema.

**The Action of Beer Yeast and the Acids that it Secretes on Diphtheria Toxin.**—Hallion[2] found that if he added a pure culture of the yeast in the must of beer to a dose of diphtheria toxin the mixture, injected under the skin of a guinea-pig, was harmless. Control animals injected with toxin and sterilized beer-must died quickly. When the toxin and yeast were injected simultaneously into different parts of the body of a guinea-pig, without having been previously mixed, the action of the toxin was not inhibited. This seems to show that the yeast neutralizes the poison directly. Further experiments led the author to the conclusion that the acidity of the culture-medium was responsible for the neutralizing action on the toxin. The experiments show that a small amount of acid is sufficient to alter the properties of a microbic toxin.

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[1] Ann. de l'Inst. Pasteur, July and Nov., 1899.

[2] Compt. rend. de la Soc. de Biol., 1899, p. 677.

**The Pancreas in Diphtheria.**—J. Girard and G. Guillain [1] have examined 29 pancreases of children dead in the course of simple diphtheria or of diphtheria associated with other diseases. There was no hemorrhagic pancreatitis found macroscopically or microscopically in any of the cases. The most striking microscopic lesions were vascular dilation. The ectasis was seen not only in the arterioles, but also in the periacinous capillaries. Endoperiarteritis and endoperiphlebitis were also found. The connective tissue of the gland was normal. The bodies of Langerhans were normal. The glandular epithelium did not present distinct lesions. Fatty change was slight; from the amount of cloudy swelling it was difficult to say whether it was pathologic or cadaveric. The ducts and the acini were well preserved. No evidences of pancreatic insufficiency were found before death; glycosuria was exceptional. The lesions are not comparable to those of the liver, the kidneys, and the suprarenal bodies found in diphtheria.

**The Relation of Diphtheria to the Conditions of the Organism.**—The predisposing causes of diphtheria are inanition, poor and insufficient food, hard labor, overcrowding, dampness, darkness, improper ventilation, noxious gases, and intercurrent diseases. According to Vallagussa,[2] animals are made more susceptible to the diphtheria poison by the ingestion of alcohol and coffee. Moderate muscular exercise, on the other hand, increases the resistance. Filtered and sterilized cultures of saprophytic germs render animals more susceptible; so do the toxins of staphylococcus, streptococcus, and Bacillus coli. The symbiosis of the diphtheria bacillus and of the streptococcus or staphylococcus favors the production of a more active toxin. Living cultures of streptococcus and staphylococcus, when inoculated simultaneously with diphtheria toxin, but not in fatal doses, are capable of producing a septicemia.

**The Frequency of the Pseudodiphtheria Bacillus in the Nasal Mucus.**—De Simoni [3] believes that the pseudodiphtheria bacillus is a common parasite in the nasal mucous membrane.

## TETANUS.

**The Pathology of Tetanus.**—Vincenzi [4] in a fatal case of tetanus studied by Nissl's method found no changes in the spinal cord and none in the gray matter of the cerebellum, the basal ganglia, or the cerebellar cortex. In the upper part of the medulla, however, he found marked changes in the large ganglion cells, particularly in the substantia reticularis and the dorsal accessory olivary nuclei. The nucleus was eccentrically placed, many of the cells were enlarged, the Nissl bodies were broken up and in some degenerated, and the nuclei in some of the cells were pyknotic; the nucleoli were not swollen, and the protoplasmic processes were not altered.

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[1] Compt. rend. de la Soc. de Biol., July 6, 1900.

[2] Ann. d'Igiene sperim., 1899, LX, 1; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[3] L'Ufficale Sanatario, No. 6, 1899; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[4] Centralbl. f. allg. Path. u. pathol. Anat., May 23, 1900.

## INFLUENZA.

**Three Cases of Extrapulmonary Localization of the Bacillus of Pfeiffer—Influenzal Pleuritis, Meningitis, Osteoperiostitis.**—H. Meunier[1] reports the case of a child, aged 20 months, who suffered from measles, followed by bronchopneumonia. Bacteriologic examination of the fluid obtained by pulmonary puncture and of blood taken from acini revealed the influenza bacillus of Pfeiffer. The bronchopneumonia became bilateral and the child died. At the autopsy a seropurulent pleurisy was found, which contained the same organism—the influenza bacillus of Pfeiffer. In the case of a child, aged 16 months, who died from convulsions on the eleventh day of a severe bronchopneumonia, autopsy showed a diffuse meningitis. Bacteriologic examination of the exudate showed the presence of the bacillus of Pfeiffer. In the case of a child, aged 6 years, influenza was followed by severe acute sore throat, which, in turn, was followed by painful swelling of the knee. On the twentieth day an operation was done and a large extra-articular collection of pus was evacuated. Trephining of a denuded portion of bone gave exit to more pus. The pus in this case contained the influenza bacillus.

**Note on a Bacillus of the Respiratory Tract and its Relations with the Bacillus of Pfeiffer.**—Elmassian[2] calls attention to the fact that he has been able to isolate from the expectoration of children and adults suffering from different diseases, such as whooping-cough, pulmonary tuberculosis, and pneumonia, a bacillus that is identical in all points with that which has been found in 3 cases of bronchial infection that clinically were of the influenzal type. The bacillus isolated presented all the characteristics assigned by Pfeiffer to the bacillus of influenza, but it developed as well upon mediums containing blood-serum as upon those without serum. On the other hand, it never grew upon ordinary peptonized gelatin. The bacillus in question does not seem to be an absolutely constant guest. The author believes that the bacillus of Pfeiffer, the rôle of which in influenza is very variable, and can not be considered to be proved, belongs to a microbic species that has a saprophytic existence on the mucous membrane of the respiratory tract similar to that of the pneumococcus. This micro-organism may multiply and become pathogenic in the course of bronchopulmonary affections.

## THE PNEUMOCOCCUS.

**The Presence of the Pneumococcus on the Normal Conjunctiva.**—Oertzen[3] found the pneumococcus in 4% of cases on the healthy human conjunctiva. The possibility of infection with the organism by way of the conjunctival sac therefore exists, but the danger of it is probably slight.

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[1] Compt. rend. de la Soc. de Biol., Jan. 12, 1900.

[2] Ann. de l'Inst. Pasteur, Aug., 1899.

[3] Klin. Monatsbl. f. Augenh., 1899, XXXVII, p. 432; Centralbl. f. Bakt., Parasit. u. Infectionskrankh., Feb. 24, 1900.

**The Presence of the Pneumococcus in the Blood.**—Pieraccini [1] found the pneumococcus in the blood in 11 out of 28 cases of pneumonia. There was some relation between the gravity of the disease and the presence of the organism in the blood.

In contradistinction to the results of other observers, Pani [2] found the diplococcus of Fraenkel in the blood in pneumonia only under special conditions, characterized by a diminution in the resistance of the organism. Their presence in the blood is, he believes, a sign of the imminence of death.

Berghius [3] did not find the diplococcus in the blood of the median vein in all cases of lobar pneumonia, whence he concludes that diplococcemia is not a constant feature in lobar pneumonia, and, moreover, bears no relation to the gravity of the disease, the splenic tumor, or the albuminuria. In the herpes vesicles complicating pneumonia he found only Staphylococcus aureus. In grave cases in which death occurred, with signs of meningism, he found changes in the cortical ganglion cells.

Silvestrini and Sertoli [4] examined the blood of 16 pneumonia cases at different periods of the disease, and found the pneumococcus 15 times. The number of bacteria in the blood often, but not always, bears a relation to the gravity of the disease.

From a study of 70 cases of infection with the diplococcus of Fraenkel, Baduel [5] concludes (1) that in the majority of cases the diplococcus is present in the blood (diplococcæmia contemporanea); (2) that in many cases it is not pathogenic for animals; (3) that when virulent, this quality usually seems to bear a relation to the gravity of the local process, for circumscribed lesions can occur with diplococci in the blood, and vice versa, and it would seem that the degree of diplococcemia is in some way connected with the resistance of the blood of the individual; (4) that virulence is usually associated with acute lesions; (5) that the persistence of the diplococcus in the blood does not constitute a complication to be dreaded, but is a common phenomenon in diplococcic infections; (6) that if the diplococcus localizes itself in the blood primarily, it may find there a favorable soil, and may then exert a pathogenic action upon the blood (primary diplococcic septicemia). This harmful action on the blood can also occur after a primary localization of the microorganism elsewhere, in which cases the diplococcemia is much more grave than in the ordinary cases.

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[1] Accad. med. Physica Fiorentina, May 3, 1899; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[2] Riforma med., Nos. 32 and 33, 1899; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[3] La Clinica med., No. 5, 1899; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[4] Riforma med., 1899, II, 41 and 42; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[5] Riforma med., 1899, I., 15; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

**The Cardiac and Muscular Lesions Produced by Pneumococcus Toxin.**—P. Carnot and L. Fournier[1] think that muscular lesions in the heart and the great vessels, in the intestines, and in the muscles of locomotion seem to be the characteristic anatomic lesions of pneumococcus intoxication, at least so far as the rabbit and the hemorrhage-producing toxin with which the authors have experimented are concerned. Pneumococcic myocarditis develops very rapidly, and, while the least movement gives rise to symptoms, when the animal is at rest very marked lesions give rise to very little trouble. Histologic examination shows a change in the transverse striations and a separation and a rarefaction of the contractile cylinders. A more advanced stage seems to be characterized by vacuolization of the cytoplasm. Frequently segmentary dissociation coexists with the lesions already described, and in 2 cases fragmentation of several of the cardiac muscle cells was present. Sometimes the diseased areas present vitreous degeneration and, rarely, the degeneration of Zenker. Pneumococcic myositis was very common in the experimental animals. In 5 cases there was complete rupture in the neighborhood of the psoas and the sacrolumbar mass, sometimes accompanied by peritoneal hemorrhage. The muscles were then extremely friable and translucent; there was often, both above and below the rupture, a punctiform hemorrhage more or less abundant. When there was no rupture, the translucent muscles, the hemorrhage, and the friability were noted, so that the muscles would break when the least traction was made upon them. Under the microscope the same lesions that have already been described in the case of the heart were found in the skeletal muscles, together with massive degeneration of the fibers and muscular phagocytosis. In the intestine, particularly in the rectum, very considerable friability was demonstrated; multiple hemorrhages showed the friability of the vessel-walls.

**The Resistance of the Pneumococcus in the Sputum.**—According to Spolverini,[2] the pneumococcus in sputum has a marked resistance, and may remain virulent from 55 or 60 to 140 days. Its degree of virulence is usually the same throughout. In the sputum are found both varieties of pneumococcus described—the edematogenic and the fibrinogenic. [These observations suggest that it is important to sterilize pneumonic sputum.]

## THE GONOCOCCUS.

**Cultivation of the Gonococcus.**—For the cultivation of the gonococcus Thalmann[3] recommends partly neutralized meat-water agar. The degree of acidity is determined with phenolphthalein as an indicator, and two-thirds of the soda solution required for complete neutralization are added. In 15 hours colonies of gonococci can be seen on this medium with the microscope. For subcultures an acid serum is better than the agar.

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[1] Compt. rend. de la Soc. de Biol., Feb. 16, 1900.

[2] Ann. d'igiene sperim., 1899, IX, 1; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[3] Centralbl. f. Bakt., Parasit. u. Infektionskrankh., June 30, 1900.

**The Culture of the Gonococcus on "Blood-gelatin."**—"Blood-gelatin" is a culture-medium described by F. Bezançon and V. Griffon [1] in February, 1899, and published in the "Comptes Rendus" for that year. They find that the gonococcus will grow on this medium in 24 hours, and that it will live for about 6 months. By the use of this medium the gonococcus was isolated from the pus of a fresh urethritis as well as from a case of acute synovitis.

Cantani [2] uses the following culture-medium: He takes human blood aseptically from a vein and mixes it with an equal volume of glycerin. This prevents clotting, and the mixture remains unaltered for a long time; from it he adds a few drops to agar or bouillon tubes, and thus obtains a favorable medium for the gonococcus. Another good culture-medium is obtained by mixing 10 cc. of ascitic serum to which a few drops of blood-glycerin mixture have been added with agar or bouillon in the proportion of 1 or 2 parts to 10. The quality of peptone used is of importance. The best is that of Merce, which is prepared from blood-fibrin.

**Staining of the Gonococcus.**—Weak solutions (1 : 10,000) of "kresylechtviolet" are recommended by Hornberger [3] for the staining of the gonococcus. In 1% solution kresylechtviolet answers well for gonococci in sections. The sections remain in the solution a few minutes, and are then transferred to alcohol and afterward to anilin-xylol (2 : 1). Amyloid, mast-cells, blood preparations, malarial plasmodia, etc., also take the stain readily.

**The Toxin of the Gonococcus.**—Panichi's [4] conclusions are as follows: (1) The changes produced in the human urethra by the gonococcus are chiefly due to the toxin elaborated by the latter. The dead bodies of the bacteria play no part at all, or only an unimportant one. (2) A urethra which reacts scarcely at all to the gonococcus is nevertheless more or less sensitive to the toxin when it is used locally.

**Gonococcemia.**—In 2 cases of gonorrheal rheumatism Panichi [5] found the gonococcus in the blood on culture.

**Pleurisy Due to the Gonococcus.**—Cardile [6] found in the fluid of an exudative pleurisy, which was the only manifestation of gonorrhea in the serous membranes, only the gonococcus; cultures were, however, not made.

**Diffuse Gonococcal Peritonitis.**—Cushing [7] describes 2 cases of acute diffuse peritonitis occurring in women and due to the gonococcus. A third is reported by Muscatello.[1] There was no autopsy, but the pus obtained from puncture of a pyosalpinx and from the peritoneum at laparotomy showed the gonococcus.

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[1] Compt. rend. de la Soc. de Biol., July 6, 1900.

[2] Riforma med., 1899, vol. I, Nos. 68, 69, 70; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[3] Centralbl. f. Bakt., Parasit. u. Infektionskrankh., April 20, 1900.

[4] Giorn. Ital. della malatt. venir e delle pelle., No. 3, 1899; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[5] La Settim. med., 1899, No. 34; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[6] La Clinica medica Ital., 1899, IX; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[7] Johns Hopkins Hosp. Bull., 1899, p. 75.

**The Streptococcus.**—Bonome and Bombicci[2] isolated from the bodies of streptococci a protein which, injected into animals, had the same action as the bodies of dead streptococci. It was possible to produce in rabbits an immunity with the protein, but only against that streptococcal species the protein of which had been used. The authors believe that there are undoubtedly different species of streptococci, differing not only in form and grouping, but also biologically. The toxic action of the protein varies with the species. The immunity indexed by protein injections is passive, and the serum of animals thus immunized has but little power.

**Beriberi.**—Fajardo,[3] of Rio de Janeiro, has discovered a blood parasite (hematozoon) in cases of beriberi. The protozoon is found in the peripheral blood and in the organs. Its presence in the brain, of which he reports 2 instances, may be of value in postmortem diagnosis. Sections are stained with methylene-blue or methylene-blue and eosin, and show pigmentary deposits in the capillaries and sometimes the parasite. He claims that in the cases studied malaria was positively excluded.

**The Etiology of Botulism.**—Van Ermengen[4] identified an anaerobic bacillus as the cause of meat-poisoning. Römer[5] was able to isolate the same organism from a ham that had poisoned a family of 4 persons. The bacillus is strictly anaerobic, from 5 $\mu$ to 7 $\mu$ long, with rounded ends, and resembles the bacillus of malignant edema. It is feebly motile, and when carefully stained retains the stain in Gram's method. Mice fed with the ham died, but bacilli could not be demonstrated in their organs. Cultures were pathogenic for guinea-pigs and mice.

**The Identity of the Bacillus Lactis Aerogenes and the Pneumobacillus of Friedländer.**—The study of the morphologic and biochemic characteristics of 4 typical specimens of Bacillus lactis-aerogenes has led L. Grimbert and G. Legros[6] to assert the complete identity of that organism with the pneumobacillus of Friedländer. The bacilli studied are immobile ; do not produce spores ; do not stain by Gram's method ; become encapsulated in pus, in blood, and in the serous exudates of inoculated animals; they are facultative anaerobic organisms; and their cultures in 3% peptone-water never produce indol. Coagulated albumin is not modified by them and milk is rapidly curdled by acidification, without alteration of the casein. The bacilli in question ferment glucose, lactose, saccharose, and dextrin, with the production of ethyl alcohol, levolactic acid, acetic acid, and succinic acid.

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[1] Il Policlinico, Aug. 15, 1899.

[2] Riforma med., 1899, I, Nos. 7 to 9 ; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[3] Centralbl. f. Bakt., Parasit. u. Infektionskrankh., Feb. 24, 1900.

[4] Zeit. f. Hyg., XXVI.

[5] Centralbl. f. Bakt., Parasit. u. Infektionskrankh., June 30, 1900.

[6] Compt. rend. de la Soc. de Biol., May 25, 1900.

**Micro-organisms in the Bone-marrow in the Course of the Infections and Intoxications in Children and in Young Animals.**—P. Haushaulter and L. Spillman [1] made cultures with the bone-marrow from the shaft of the tibia in 49 children between the ages of several months and 2 years. The children had died of bronchopneumonia, 37; gastro-enteritis, 5; erysipelas, 1; miliary tuberculosis, 2; and infantile cachexia, 4. In the majority of cases simultaneous cultures were made from the spleen. The cultures gave positive results in 12 cases and negative results in 37 cases. Bacillus coli communis was found in 4 cases of bronchopneumonia; the streptococcus, the staphylococcus, and the pneumococcus were found in 3 cases of bronchopneumonia. [Whether one organism in one each of 3 cases or all 3 organisms in all 3 cases is not stated.] In 2 cases of bronchopneumonia and in 1 case of gastro-enteritis the cultures showed a special organism, for which the name of enterococcus is suggested. In the 12 cases, 9 cultures from the spleen were made, of which only 2 were positive; each time the colon bacillus was the offending organism. Out of the 37 sterile cases, 33 cultures were made from the spleen; and among these, a positive result was obtained but once, and here again the colon bacillus was found. Inoculation experiments were made from the bone-marrow of young animals in 44 cases. Of these, 27 cases were sterile and 17 gave positive results. The experiments seem to show that there is nothing to indicate in advance whether or not the marrow is affected.

**The Rôle of Habit in the Determination of Bacterial Localization.**—Bezançon and Abbé [2] state that a micro-organism that has resided in a tissue, and that has determined a pathologic lesion in that tissue, acquires by that fact a certain tendency to localize itself again in a similar tissue. By experiment they found that a staphylococcus obtained from a purulent human arthritis, in spite of successive inoculations, always retained its property of localizing in the articulations; while a staphylococcus that, in one of the rabbits used for the experiments, had passed into the heart's blood lost this property of localizing itself on the joints and gave rise only to suppurating lesions of the viscera or to a septicemia. It would seem that the growth of a micro-organism in an organ accustoms it, the bacterium, to defend itself against the phagocytes or makes it tolerant to the action of the humors of a particular tissue, so that it acquires special qualities of resistance toward that tissue which enable it to localize itself in that to the exclusion of other tissues. [Pfaundler has already shown that identical bacteria inoculated into animals of the same species become modified so that afterward on being recovered they will be different in certain respects. It is not improbable, as shown by the foregoing experiments, that this metamorphosing influence extends even to the different tissues of the body.]

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[1] Compt. rend. de la Soc. de Biol., Jan. 26, 1900.

[2] Compt. rend. de la Soc. de Biol., Jan. 19, 1900.

**The Bacterial Content of Market Milk.**—The studies of Beck [1] had for their object (1) the determination of the presence of bacteria, particularly of tubercle bacilli, in milk; (2) to find out whether the destruction of these bacilli, particularly the tubercle bacilli, is possible by a single boiling, or whether prolonged boiling is necessary; (3) to learn which of the kitchen utensils is best adapted for the boiling of the milk. (1) He found that only about one-fifth of the specimens of Berlin milk examined was free from pathogenic germs. He found streptococci in 62% and tubercle bacilli in 30%. (2) A single boiling of the milk is insufficient to destroy all the germs. The streptococci, it is true, are killed thereby, but to annihilate the tubercle bacilli a 3-minutes' boiling is required. To prevent the boiling over or the scorching of the milk, it has to be stirred from the moment it begins to boil. (3) Earthen vessels are best suited for the purpose of boiling the milk. [These researches are of great importance, and the results should be widely known. They render it very questionable whether the pasteurization of milk destroys tubercle bacilli.]

**Micro-organisms in the Mouth of the New-born.**—Giuseppe Campo [2] has carried out a series of researches in order to ascertain (1) whether the mouth at birth is free from germs; (2) the appearance of germs along with the establishment of the mammary function of life; and (3) their pathogenic action. The contents of the mouth were taken at the moment of birth, while the head was still on the perineum; 4 hours later, when respiration was going on; and 24 hours later, when lactation had begun. Ten infants were examined at these three times, and 21 others at one or other of the times named. Campo concludes that the mouth is sterile at the time of birth. Although germs were found in some of the cases, sources of contamination existed; in some cases, for example, the material was collected by midwives. The effect of respiration was an immediate entrance of germs into the mouth. Those found were the bacteria of the air. They were Bacillus mesentericus vulgatus, Bacterium termo, and Bacillus ulna. The first effect of lactation was found to be a diminution in the number of germs, possibly due to the mother's milk washing them down into the gastrointestinal tract; but its second effect seemed to be to increase the number of kinds of germs. The organisms found were, in the order of frequency: Bacillus mesentericus vulgatus, Bacterium termo, Bacillus ulna, Bacillus subtilis, Bacillus lineola, Bacillus leptothrix, and 3 unidentified germs. None of them had any pathogenic power.

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[1] Deut. Vierteljahrsschrift f. öffentliche Gesundheitspflege, 1900, p. 430; Centralbl. f. Bakt., Parasit. u. Infectionskrankh., 1900, XXVIII, 452.

[2] Pediatria, VII, 229, Aug., 1899; Brit. Med. Jour., Oct. 21, 1899.

**Bacterial Findings in Cadavers.**—Löw [1] examined 112 cadavers, studying particularly the urine, blood, and renal pelvis. In 109 cases bacteria were found in the bladder 43 times, 31 giving the colon bacillus. The bacteria were rare in autopsies made 10 hours after death; in those made 1 or 2 days postmortem, half gave positive results. All the cases in which toward the end of life catheterization had been done gave positive results; a single catheterization toward the close of life, even with the most careful aseptic precautions, usually was sufficient to infect the urine. In 83 cases in which the renal pelvis was examined positive results were obtained in 14; and of 45 examinations of the blood from the vein of the arm, 7 gave bacterial cultures. Löw does not believe that the presence of bacteria was, as a rule, the result of postmortem invasion. In some experiments made on animals in which poisonous cultures were injected into the peritoneum, positive results were obtained only when the bacteria were injected into the intestine after a laparotomy. He concludes that postmortem emigration of bacteria from the intestine to the neighboring organs—liver, gall-bladder, and urinary bladder—may occur, but a dissemination to the heart or the circulation is not likely. The positive results found in cadavers are usually due to infection *intra vitam* or in the death agony; a postmortem multiplication of bacteria is, however, possible. If experiments with rabbits and mice give different results, it is because the behavior of the intestines of these animals toward the transmigration of bacteria is different from that of the human intestine.

**Production of Fat by Bacteria.**—By means of sudan III, Sata [2] has shown that a number of bacteria produce fat, and has studied this process particularly in the actinomyces; the anthrax, the glanders and hay bacilli, and Staphylococcus aureus form fat only on glycerin-agar—not on agar without glycerin.

**Thermic Phenomena Presented by Bacterial Cultures.**—Giuffre [3] has made calorimetric studies of cultures of various bacteria, and finds that from this point of view bacteria are divisible into 3 groups: The first group comprises those which from the beginning of their growth produce heat; the second, those which abstract it from their surroundings; the third, those which in the beginning abstract it and later give it off again. These facts permit of their classification into 2 great divisions—the thermogenic and the algogenic. The importance of these facts for the question of the mechanism of fever is evident.

**Staining of Bacteria in Sections Treated by Van Gieson's Method.**—The following method is proposed by Dreyer [4]: The sections are hardened in formalin, embedded in paraffin, affixed to the slide with 30% alcohol, and freed of paraffin. They are then treated as follows: (1) Watery solution of methyl-violet (about 1 %) or gentian-violet, from

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[1] Zeit. f. Heilkunde, Bd. XXI, Heft 1.

[2] Centralbl. allg. Path. u. pathol. Anat., Feb. 15, 1900.

[3] Tenth Cong. Ital. Soc. for Intern. Med.; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[4] Centralbl. f. Bakt., Parasit. u. Infektionskrankh., April 20, 1900.

3 to 5 minutes; (2) washing in distilled water; (3) concentrated watery solution of picric acid, from 3 to 4 minutes; (4) drying with filter-paper; (5) anilin oil to which 0.1 % picric acid is added; the sections remain until they are grayish-yellow and no longer give off a violet color; (6) careful washing in distilled water; (7) Delafield's hematoxylin, from 5 to 8 minutes; (8) washing in distilled water for 5 minutes; (9) acidulated picric-acid-fuchsin (2 or 3 cm. picric-acid-fuchsin to which 1 drop of a 1 % solution of glacial acetic acid is added), from 3 to 5 minutes; (10) washing and dehydrating in absolute alcohol from ½ to 1 minute; (11) xylol; xylol-damar. This stains practically all bacteria except those decolorized by Gram's method; the tubercle bacillus is especially well stained. For the last-named bacillus, for yeasts, etc., it is well to prolong the first step to ½ or 1 hour in the thermostat at 37° C.

**An Apparatus for the Preparation of Roll Cultures.**—Nuttall[1] describes a simple and efficient apparatus for the preparation of Esmarch's roll cultures. It consists of a block of marble in which grooves are made for test-tubes; the test-tubes are laid in these grooves and water is allowed to trickle upon them.

Sata[2] recommends the following method for staining actinomyces in sections with sudan III; (1) Fixation in formalin; (2) washing in water; (3) sectioning with the freezing microtome; (4) feeble hematoxylin staining; (5) a few minutes in alcohol; (6) from 12 to 24 hours in a saturated alcoholic solution of sudan III; (7) washing in alcohol; (8) embedding in glycerin.

**Immunity; Toxins; Antitoxins; Agglutins.**—Wassermann[3] distinguishes two kinds of serum: (1) antitoxic, which acts upon the bacterial toxins and is not bactericidal; (2) bactericidal, which destroys the bacteria, but has no action upon the toxins. An animal may be immunized with large quantities of antitoxic serum, and may yet succumb to bacterial infection. The peritoneal exudate of the animal may be utilized to immunize another animal. Two substances are concerned in immunity: (1) an immune body or intermediate body, and (2) a complement or end body. The latter is a ferment, capable of dissolving the bacterial cell. The intermediate body serves to attach the end body to the bacterial cell. It is never present in the normal body and is developed only during infection. For the cure of infection sufficient quantities of the intermediate and end bodies are necessary. Up to the present, attention has been paid only to the intermediate body. If normal serum and immune serum are simultaneously injected into rabbits, the animals may be protected against typhoid infection. Ehrlich has stated that it is difficult to find complement bodies to the immune bodies. The object should be to discover a complement which would be stable in the human organism and would not be destroyed by the living cells. Perhaps monkeys could best be used for the preparation.

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[1] Centralbl. f. Bakt., Parasit. u. Infektionskrankh., May 4, 1900.

[2] Centralbl. f. allg. Path. u. pathol. Anat., Feb. 15, 1900.

[3] Eighteenth Cong. for Intern. Med., April 18–21, 1900; Centralbl. f. allg. Path. u. pathol. Anat., May 23, 1900.

**Racial Immunity.**—Prettner [1] found that buffaloes were not susceptible to experimental tuberculosis infection.

**Contribution to the Study of Immunity.—Properties of Mixtures of Toxins with Their Antitoxins.—Constitution of Toxins.**—Danysz [2] concludes (1) that the particular method of the action of toxins, as well as the properties of mixtures of toxin and antitoxin, do not depend upon a differentiation of the toxin into different substances more or less toxic, as pointed out by Ehrlich, but simply upon the presence of phosphates in the mixtures in greater or less proportion. (2) That according to the proportion of phosphates and other salts contained in the mixtures (and in the tissues, when the action of a toxin upon a living organism is considered), the same active substance may produce variable effects, and in the same way the difference of the sensibility to the action of a toxin upon animals of a different species may be accounted for.

**Immunity Against Malarial Infection.**—Celli's observations [3] show that certain human beings are immune to malaria, even in the most malarious of districts. This immunity may even extend to experimental infection. Sometimes it is innate; in other cases it is acquired through diseases, chiefly paludal cachexia, and also after infections of short duration. This immunity is not so prolonged as the congenital. Nothing was found in the blood-serum of either the immune or the convalescent to indicate that recovery and immunity are dependent upon qualities of the serum. It was also not possible to immunize human beings with the blood and tissue-juices of animals immune to malaria. Extracts made from various forms of anopheles and culex gave negative results. Quinin is also inactive in protecting human beings against experimental malaria. Euchinin and methylene-blue have some prophylactic value. Although the clinical symptoms of malaria are strongly suggestive of the action of a toxin, Celli's experiments failed to reveal the presence of such a substance, as well as of an antitoxin.

**The Influence of Castration on Resistance to Infection.**—Sirleo [4] found that castration, both in male and female animals, lessened the resisting power against infection.

**The Protective Action of the Peritoneum in Infections.**—It has been shown by Binaghi [5] that a slight alteration in the peritoneum permits the intestinal bacteria to enter the abdominal cavity, even if the intestinal epithelium is uninjured. This shows that the protective action of the intestinal epithelium is supported by the resisting power of the peritoneal endothelium.

**The Protective Rôle of the Liver and the Lung.**—The lung is usually considered to be a simple filtering membrane the function of which it is to make sure the interchange of gases. The studies made by Roger [1] lead to the conclusion that the lung is an organ that has a protective power against certain infections and intoxications. This power is already well known as one of the characteristics of the liver and the kidneys.

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[1] Centralbl. f. Bakt., Parasit. u. Infectionskrankh., 1900, XXVII, 3.

[2] Ann. de l'Inst. Pasteur, July, 1899.

[3] Centralbl. f. Bakt., Parasit. u. Infectionskrankh., 1900, XXVII, 3.

[4] Tenth Cong. della soc. Ital. di med. interna.; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[5] Riforma med., 1899, IV, Nos. 37 to 39; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

**Antitoxic Globulins.**—Atkinson,[2] under the direction of Park, has made a study of the globulins of diphtheria-antitoxin horses. The globulin of the horse serum could be separated into 5 fractions, each one of which had antitoxic properties; and the total amount of globulin appeared to contain practically all the antitoxin of the serum. Park intends to employ the antitoxic globulins instead of the antitoxic horse serum, in the hope of avoiding the rashes so common after the injection of the latter.

**Bacteriolysins.**—N. Gamaleia [3] defines bacteriolysins as complex substances containing a peptic ferment, combined with a bacterial derivative. The latter constituent confers upon them their specificity—the power of dissolving certain bacteria; while the former constituent explains the digestive phenomena of their action. For the preparation of certain lysins the peptic ferment has to be added in excess; for other bacteriolysins this peptic derivative is furnished by the bacteria themselves. Bacteriolysins act not only in distilled water, but also in the blood-serum; even when injected into the economy, they continue to exert their specific action; this has been proved in connection with the bacteriolysins of the tubercle bacillus, bacteriolysis taking place when the ferment is introduced beneath the skin of the guinea-pig and the tubercle bacillus in the peritoneum. The conclusion follows that artificial bacteriolysins carry specific bactericidal action into the animal economy. The bacteriolysins explain the destruction of micro-organisms in the animal body and natural and acquired bacterial immunity. The author has shown that while the bacteriolysins destroy the bacteria in the animal body, they allow the toxins to escape. The latter substances are difficult to demonstrate by any known means.

**A Study of the Relations between the Agglutins and the Lysins in Anthrax.**—The agglutinating property of the serum of animals inoculated with anthrax, according to O. Gengov, [4] is a specific property. The agglutins of anthrax do not seem to pass to the fetus in immunized animals. The agglutins may pass through the walls of the vessels into the body-fluids, but they do not pass in their totality. They dialyze perfectly when the inferior liquid is distilled water, although they pass in greater proportion when the inferior liquid approaches chemically the normal serum of the dog. On the other hand, they will not pass through a collodion sac placed in the peritoneal cavity of an immunized animal. The agglutins of anthrax toward heat behave as do all other agglutins. There are no bactericidal substances in the normal serum of the dog, nor is the specific agglutinating serum bactericidal. Consequently agglutins and lysins are different substances. The living leukocytes are by no means the producers or the retainers of the agglutins, and their death does not result in the passage of bactericidal substances into the serum, where they exist primarily. It is difficult to attribute to the organs or to the cells of the organism any intervention whatsoever in the manufacture of agglutins; on the contrary, the organism seems to be passive. In anthrax, at least, the phenomenon of agglutination appears to have no relation to immunity nor to the defense of the organism.

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[1] Compt. rend. de la Soc. de Biol., 1899, p. 213.

[2] N. Y. Path. Soc., Oct. 11, 1899; Med. Rec., Nov. 18, 1899.

[3] Compt. rend. de la Soc. de Biol., 1899, p. 158.

[4] Ann. de l'Inst. Pasteur, Aug., 1899.

**The Agglutination of Bacillus Anthracis by Normal Human Blood.**—Lambotte and Maréchal [1] have succeeded in producing agglutination phenomena in cultures of Bacillus anthracis by the addition of normal human serum in as high dilution as 1 : 500. The serum was obtained from a woman suffering from advanced tuberculosis. In many other persons not suffering from anthrax the serum has been found to produce agglutination of the bacillus in dilutions varying from 1 : 50 to 1 : 300. In 4 children, aged respectively 12 hours, 1 day, 3 days, and 8 days, the serum agglutinated the anthrax bacillus in dilutions of 1 : 50, 1 : 40, 1 : 60, and 1 : 50, respectively.

**On the Presence of Specific Agglutins in Bacterial Cultures.**—The studies of Malvoz [2] have led him to conclude that it is not necessary to assume the existence of normal or pathologic secretions, due either to the infection itself or to cellular processes of immunity, in order to explain the agglutinating action of serum. The specific agglutins may be found in the bacterial cultures themselves, at least so far as anthrax is concerned; and these, added to new emulsions of the bacilli, produce the characteristic clumping. The author has succeeded in producing clumping of the anthrax bacillus by the addition of ordinary bouillon to the hanging-drop preparation and by the addition of bouillon in which the anthrax bacilli have been grown and from which the organisms have been removed by the centrifugating apparatus. The author believes that the bacilli carry with themselves certain products of which the nature is unknown, and which, in the presence of substances like bouillon, result either in the formation of a coagulum that incloses the micro-organisms or in a special viscosity of the rods that causes them to adhere to each other. The mechanism of agglutination is not known. The author explains the agglutinating property of the serums of immunized animals by assuming that repeated inoculations with microbic products surcharge the blood with the substances manufactured by the bacilli and found in cultures. When the blood is drawn and placed in relation with homologous organisms, agglutination is determined.

**On the Mechanism of the Agglutination of Micro-organisms by Normal or Immunized Serums.**—After examining the theory of

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[1] Ann. de l'Inst. Pasteur, Aug., 1899.

[2] Ann. de l'Inst. Pasteur, Aug., 1899.

Paltauf and Nicolle, and that of Bordet, Arloing [1] concludes that of the theories advanced up to the present time to explain the agglutination of micro-organisms, the one that connects the reaction with the modification of the molecular attractions under the influence of the serum is the most reasonable. But it is impossible to accept this hypothesis unreservedly, particularly in the shape in which it is supported to-day, because it does not adapt itself exactly to all the observed facts. The part played by the changes in the micro-organisms and that played by the modifications of the surrounding mediums need more careful study.

**Serum Reaction in Proteus Vulgaris.**—Rodella [2] has made a series of experiments which leads him to the following conclusions: Agglutination of Proteus vulgaris occurs in the blood of guinea-pigs after inoculation with virulent cultures, with dead cultures, and with filtrates; also after feeding cultures to guinea-pigs. The blood of newly born guinea-pigs, the offspring of mothers that had been inoculated from 20 to 39 days before with proteus cultures, possessed agglutinating properties. An animal that had been inoculated 10 days previous to the birth of the young did not transmit the agglutinating reaction. The milk of guinea-pigs the young of which gave a positive reaction was also agglutinating. Thread formation was also observed, but was not constant. As Proteus vulgaris is acquiring considerable importance in human pathology, the discovery of its agglutination by the serum of infected animals is important. The organism has been found in some cases of ulcerative endocarditis, and it might be worth while, in instances of this disease in which the etiology is obscure, to try the effect of the serum on cultures of proteus.

**The Nature and Action of Snake-venom.**—Fraser has found that serpents' venom introduced by the mouth is harmless. The facts that render it innocuous were not determined by him except to the extent of proving that bile is antitoxic. R. H. Elliott [3] has made some experiments to discover the cause of immunity to the introduction of cobra venom by the mouth. He found that when the venom was given to dogs after the side-tracking of the biliary secretion by means of a continuous fistula, the animals nevertheless survived, so that the bile can not be the cause of the immunity; that immunity could not be due to inability on the part of the intestine to absorb venom, for he showed that the direct introduction of venom into the intestine was speedily fatal. He then made an artificial anus in such a way as to prevent the peptic, pancreatic, and biliary secretions from passing into the lower segment. When the venom was introduced into the latter segment, it promptly produced a fatal result. He concludes from these experiments (1) that cobra venom can be absorbed through the walls of the small intestine, but not so readily as through the channels of the subcutaneous tissue; (2) that some change or changes are wrought in swallowed cobra venom before it has time to reach a rapidly absorbing area. Experiments with the pancreatic ferment trypsin showed that it has an inhibitory action on the poison, and that its properties explain readily why venom fails to kill when swallowed. It was also found that when animals did not die after swallowing poison, some change took place in them which rendered them more susceptible to the influence of poison after it was introduced subcutaneously. If time is given, the effects of swallowed poison are in the direction of immunization; but in the mean time they lessen the animal's resistance to venom.

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[1] Compt. rend. de la Soc. de Biol., 1899, p. 40.

[2] Centralbl. f. Bakt., Parasit. u. Infektionskrankh., May 4, 1900.

[3] Brit. Med. Jour., May 12, 1900.

## TUMORS.

**The Etiology of Tumors.**—Schüller [1] has isolated from sarcomas and carcinomas a round or oval vesicular parasite, larger than a red corpuscle, refractive, and golden yellow or brownish in color. It consists of a resistant envelope of light color and dark granular contents. In the hanging drop the young forms seem to possess at the periphery fine processes resembling cilia. The organisms are also demonstrable in the tissue of the tumors, particularly when alum-carmin or alum-hematoxylin was used for staining. As a culture-medium Schüller employed the tumor tissue itself, keeping it in sterile, closed glass dishes in the dark at body-temperature. Animal experiments have been made, but are not far enough advanced to be reported. The organisms multiply by fission and by budding. [Why the author believes these bodies to be protozoa is not readily apparent. If parasites at all, they are probably blastomycetes, the relationship of which to malignant tumors is becoming more and more doubtful.]

**Myxomycetes as the Producers of Tumors in Animals.**—In a preliminary communication Podwyssotzki [2] describes the results produced in animals by the inoculation of a plant parasite—the Plasmodiophora brassicæ. In rabbits and guinea-pigs tumors were produced, consisting of mesodermic cells, resembling sarcoma or endothelioma or the granulomas of the leprosy type. The fixed cells, particularly the endothelia of the perivascular space, were involved, giving rise to what the author calls a myxomycetic perithelioma. Leukocytic infiltration is present in the earlier stages, but afterward disappears. The tumor-cells contain spores of the plasmodiophora; the older the cells, the more numerous are these spores. Giant cells are present in places. The presence of karyokinetic figures in some of the spore-containing cells proves that the spore can induce cell multiplication. The appearance of the cells suggests active phagocytosis. The spores in the protoplasm of the cells are surrounded by minute fat droplets, and such cells appear to consist of a large number of vacuoles, each vacuole being surrounded by fat droplets. In a very malignant sarcoma of the abdomen, kidney, thyroid, and other organs, in a child, the author found similar large, vacuolated, fatty cells. Minute inclosures were found in the vacuoles. The phagocytosis in the tumors produced by the plasmodiophora in guinea-pigs and rabbits seemed to be sufficient to arrest the growth of the tumor and to prevent its becoming malignant. [As no illustrations are given, we must wait for the author's final report before forming a definite judgment concerning his alleged discovery ; but even if the parasite produces a formation of new tissue, it may be only a peculiar form of granulation tissue, far removed from a true malignant tumor.]

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[1] Centralbl. f. Bakt., Parasit. u. Infektionskrankh., April 20, 1900.

[2] Centralbl. f. Bakt., Parasit. u. Infektionskrankh., 1900, XXVII, 3.

**Blastomycetes in Sarcoma.**—Biagi [1] isolated from a sarcoma of the patella a blastomyces differing from those hitherto described and having marked pathogenic properties. He attributes to the blastomyces a rôle, but not the sole one, in the production of the tumor.

**Blastomycetes and Tumor Formation.**—Peterson and Exner [2] repeated Sanfelice's experiments with Saccharomyces neoformans and Saccharomyces hominis. The animals all became ill and died of cachexia, and the autopsy showed an enormous distribution of the yeast-cells with a minimum reaction of the tissues, tumor-like formations at the site of injection, and in 3 cases tumors at a distance. All the tumors consisted of granulation tissue, and resembled actinomycosis much more than true tumors ; whence the authors conclude that definite proof of the relation of saccharomyces to tumor-formation has not been brought.

Carini [3] was unable in 14 specimens of tumor which were removed during life and kept aseptically to demonstrate the blastomycetes culturally ; while in 6 of them the histologic examination apparently showed the presence of these organisms. Only 1, a cystopapilloma exposed for several hours to the air, furnished colonies of blastomycetes. He was never able to produce new growths by the inoculation of blastomycetes. In a large number of specimens of tumors stained by specific methods the blastomycetic forms of Sanfelice were found. These same forms are sometimes found in tuberculous and healthy organs.

**Amitotic Cell-division in Pathologic New Formations, Particularly Sarcomas and Carcinomas.**—Nedjelsky,[4] after a résumé of the literature on cell-division, with particular reference to the so-called direct division of cells, describes observations made by him on a large number of malignant tumors. Former writers on the subject had entirely overlooked the importance of the nucleolus in amitotic cell-division, and it is the changes taking place in it that were particularly investigated. Remak had pointed out that amitotic division began with cleavage of the nucleolus. The same view had been held by Virchow, while Ranvier and Arnold believed that it commenced in the nucleus. Under pathologic conditions, particularly in tumors, the pro-

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[1] Il Policlinico, No. 10, 1899 ; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[2] Beiträge zur klin. Chir., Bd. XXV, 769 ; Centralbl. f. allg. Path. u. pathol. Anat., June 15, 1900.

[3] Bull. soc. med. e chir. di Pavia, No. 7, 1899 ; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[4] Beiträge zur pathol. Anat. u. allg. Path., 1900, Bd. XXVII, Heft 3.

# EXPLANATION OF PLATE 3.

Fig. 1.—*Carcinoma of ovary:* The nucleus is enlarged and the nucleolus visible, the volume of the latter being increased preparatory to division; also a nucleolus which is dividing and some which have already divided. Fig. 2.—Same tumor: Division of nucleus and protoplasm; a portion of the dividing nucleus is engaged in dividing again. Fig. 3.—Same tumor: Simultaneous division of all constituents of the cell. Fig. 4.—*From the cartilaginous portion of an osteochondrosarcoma of the tibia:* Simultaneous division of all cell constituents. Fig. 5.—*Carcinoma of the ovary:* Early stages of the division of the cell and protoplasm; the nucleolus has been torn across into two parts. Fig. 6.—Same tumor: All parts of the cell have been completely divided. Fig. 7.—*Carcinoma of the upper lip:* The nucleus repeats in its division the form of the nucleolus; the protoplasm does not divide. Fig. 8.—*Carcinoma of the ovary:* All parts of the cell divide. Fig. 9.—Same tumor: The nucleus is completely divided; the protoplasm and the nucleolus are approaching completion of division. Fig. 10.—Same tumor: In an undividing protoplasm there are two nuclei, of which the one divides into three, and the other into two parts. Fig. 11.—Same tumor: Protoplasm and nucleus do not divide. Fig. 12.—Same tumor: Protoplasm does not divide; the nucleus consists of three parts, of which the first has separated itself completely and contains the fusiforn nucleolus. Fig. 13.—Same tumor: Protoplasm does not divide; the nucleus is elongated and the nucleolus divided into three parts. Fig. 14.—Same tumor: The protoplasm does not divide; the nucleus has in its middle portion a rod-shaped nucleolus, the ends of which are fused with the nuclear membrane. Fig. 15.—Same tumor: Simultaneous division of all parts of the cell. Fig. 16.—*Sarcoma of the uterus:* The nucleus is elongated and the chromatin is heaped up at the ends; the nucleolus has divided. Fig. 17.—*Sarcoma of the orbit:* Three cells, in various stages of nuclear division by the mode of stretching; in two cells the protoplasm is engaged in dividing. Fig. 18.—*Sarcoma of the uterus:* The nucleolus has divided; the nucleus divides by stretching into two parts; beginning division in the protoplasm. Fig. 19.—*Sarcoma of the uterus:* Marked polymorphism of the nuclei: nucleus *a* divides longitudinally by constriction; *b*, similar division with a broader constriction; *c*, division of all parts of the cell; *d*, nucleus divided into two unequal parts, the smaller of which is again dividing; *e*, as in *d*, only that a part is dividing into three fragments, which are united to the remaining mass by fine threads; *f*, the nucleus contains a nucleolus; *g*, from the large nucleus a smaller portion is constricted off—a future so-called paranucleus. Fig. 20.—*Sarcoma of the uterus:* The parts of the dividing nucleus are united by threads. Fig. 21.—*Sarcoma of the uterus:* The protoplasm divides into two parts and the nucleus into two unequal parts, the larger of which is again dividing into three. Fig. 22.—*Sarcoma of the uterus:* The nucleus has a branching form. Fig. 23.—*Sarcoma of the uterus:* Another stage of nuclear division. Fig. 24.—*Sarcoma of the uterus:* The parts of the dividing nucleus are united by bands. Figs. 25, 26, 28.—*Sarcoma of the orbit:* Beginning division in the length of the nucleus. Fig. 27.—*Sarcoma of the orbit:* Deep constriction, transverse to the nucleus. Figs. 29, 30, 31.—*Sarcoma of the orbit:* Several indentations on the surface of the nucleus; different stages of division. Fig. 32.—*Carcinoma of the ovary:* The nucleus is enlarged and shows shallow indentations; the nucleoli are divided. Fig. 33.—*Carcinoma of the ovary:* The indentations are more marked than in figure 32; the nucleoli are very large and contain several shining granules. Fig. 34.—*Carcinoma of the ovary:* All parts of the cell are dividing. Fig. 35.—*Sarcoma of the uterus:* Division of the nucleus into several parts. Fig. 36.—*Carcinoma of the ovary:* The protoplasm is completely divided; the nucleus is dividing. Fig. 37.—*Carcinoma of the penis:* On the surface of the nucleus there is a spheric excavation with a nucleolus; in the protoplasm near the nucleus there is a nucleus the size of the excavation—a so-called paranucleus. Fig. 38.—*Sarcoma of the uterus:* The nucleus shows several indentations on its surface and a paranucleus in the protoplasm. Fig. 39.—*Sarcoma of the uterus:* The nucleus is divided into two parts; the nucleolus is also divided. Fig. 40.—*Sarcoma of the uterus:* Nucleus divided into three parts; the constriction in the protoplasm corresponds to the points of division of the nucleus. Fig. 41.—*Sarcoma of the uterus:* Similar to the foregoing. Fig. 42.—*Several cells from a cystadenoma of the ovary:* Constriction of the nuclei in the transverse axis. Fig. 43.—*Sarcoma of the uterus:* Mononuclear giant cell. Fig. 44.—*Carcinoma of the ovary:* A mononuclear giant cell.

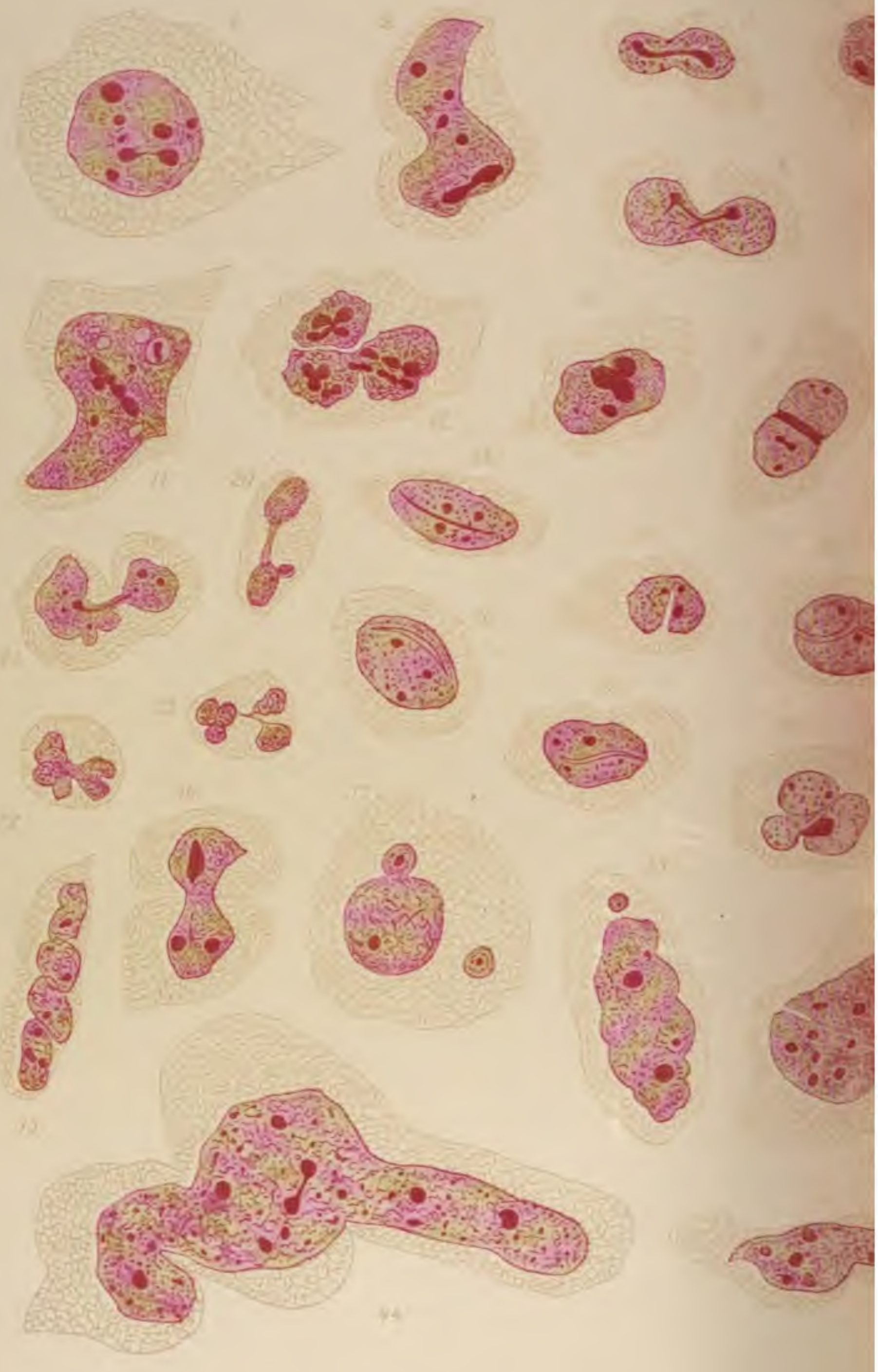
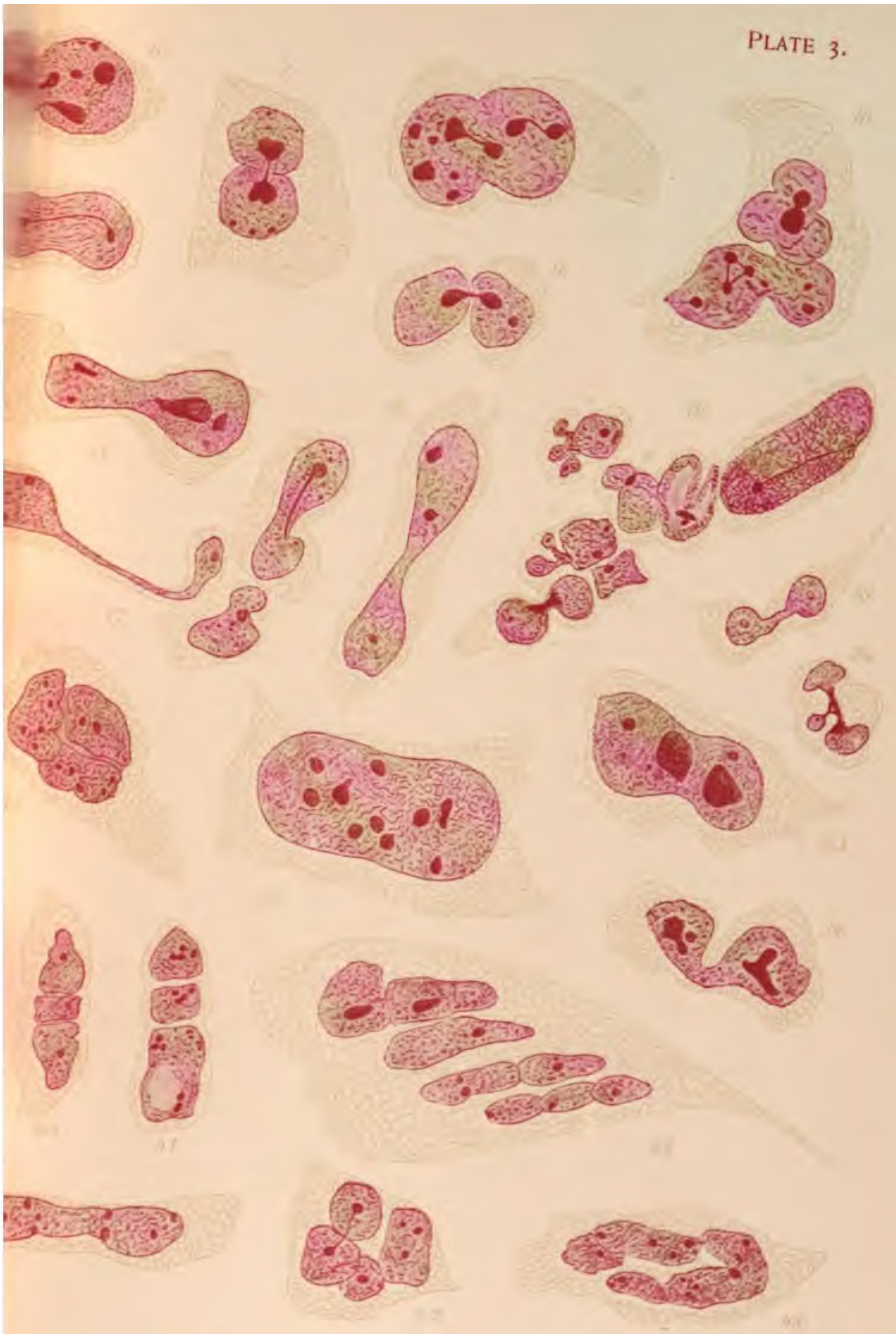


PLATE 3.



cess may be modified, and Nedjelsky describes 6 modes of what might be called abnormal division : (1) The nucleolus divides, while the nucleus and protoplasm show no trace of division (plate 3, Fig. 1). (2) The nucleolus and nucleus divide, but the protoplasm remains inactive (Fig. 7). (3) The nucleoli and protoplasm divide, while the nucleus lags behind (Fig. 36). (4) The nucleus is divided, while the nucleolus and protoplasm are not (Fig. 9). (5) Before the division of the nucleus is completed, one of its parts already begins to divide (Figs. 19, 20, 21). (6) A part of the dividing nucleus is large ; another, extremely small (Figs. 37 and 38). In neither sarcoma nor carcinoma is any peculiar and exclusive form of amitotic division discoverable, and in all pathologic processes various forms of amitotic cell-division occur. In some of the tumors, particularly in an ovarian carcinoma, the author found both mitotic and amitotic cell-division. Various types of pathologic karyokinesis were discovered in the tumors studied. In some nuclei the loops of chromatic substance varied in thickness, as if some had not divided. In others the chromosomes of the equatorial zone had variable thickness and length. In others there was a delay in the migration of the loops toward the poles, so that in one pole there was a complete aster, while in the other only a few chromosomes had arrived. Displacement of the chromosomes outside of the nucleus was also found. A polymorphism of the nucleus is quite constant in sarcomas and carcinomas ; this polymorphism is usually, but not always, a sign of degeneration on the part of the nucleus. Amitotic division begins with an active enlargement of the nucleolus. The latter becomes elongated and thinner at the middle, and gradually is torn across. In other cases, instead of an abrupt tear, the middle portion becomes indented or constricted. Sometimes several of these constrictions are seen, and in that case the nucleolus is divided into several new nucleoli. The division of the nucleolus is connected with that of the nucleus, the process, as a rule, being that the nucleolus lies at right angles to the long axis of the nucleus, and approaches the walls of the latter, as it elongates, eventually fusing with it. Nedjelsky calls the first division described that by stretching ; and the other, that by constriction. The division of the nucleolus is soon succeeded by that of the nucleus, the division of the latter being also inaugurated by an increase in size. Afterward the nucleus becomes elongated, the chromatic substance accumulating at the ends, and eventually it is torn across at its middle. The newly formed nuclei, each containing its nucleolus, are either equal or unequal. Instead of being torn across in the manner described, the nucleus may divide by constriction. Cylindric epithelium generally divides by the method of constriction.

*The Origin of Giant Cells.*—The important feature of the giant cell is the size of the cell, and not so much the number of nuclei. Three types of cells may be recognized : those with simple, those with compound, and those with multiple nuclei. They can originate by mitotic or by amitotic cell-division. In no instance was the author able to find in tumors any evidence that giant cells had been formed by fusion of other cells. Giant cells are much more frequently formed by amitotic than by mitotic division. Division of the protoplasm after amitotic nuclear division frequently occurs in malignant tumors. The mechanism of such division does not differ from that in karyokinesis. In cirrhosis and carcinoma of the liver evidences of amitotic cell-division were also found in the hepatic cells, but division of the protoplasm very seldom occurred. In the kidney in interstitial inflammation and chronic congestion amitotic cell-division was found, but was rare. Division of the protoplasm was never observed. The final conclusions of the author are as follows: (1) Amitotic division occurs in the cells of new growths and in the cells in other pathologic processes. (2) Division begins with enlargement of the nucleolus or nucleoli, this enlargement being much less marked in connective-tissue tumors than in epithelial tumors. (3) Division proceeds either by constriction or by stretching, or by both. (4) Division of the protoplasm occurs more frequently than has been assumed. (5) In the formation of giant cells division occurs in the same manner as in other cells. (6) Amitotic division participates actively, just as mitosis, in regeneration.

**Special Evolution of the Attraction Sphere in the Cancer-cell.** A. Borrell [1] believes the bodies described by Sawtchenko in carcinoma cells are rapidly developed attraction spheres which have evolved in a special manner. He bases his opinion on the similarity in color reaction when the same method is applied to carcinoma cells and to the spermatic cells of the guinea-pig.

**The Presence of Fat in Endothelioma (Perithelioma) of Bone.**—Ritter,[2] in a central tumor of the tibia consisting of ramifying and anastomosing capillaries, with a mantle of large and multinuclear cells, found abundant glycogen in the cells, and in some of them also a finely granular fat. As the cells containing the fat were in no way degenerated, Ritter believes that the fat-formation was a sort of secretion, analogous to that of the glycogen, and not a sign of degeneration.

**Intravascular Growth of Endothelioma.**—MacCallum [3] describes a most remarkable growth arising from the left testicle of a man aged 28. The organ was removed, but the tumor developed in the veins, extending along the spermatic into the great abdominal veins, thence sending metastases to the lungs and other organs. It did not arise from the epithelium of the testis, but from cells lining clefts, probably lymph-spaces. The tumor formed papillary masses which burst into the veins, being there covered by endothelium in the manner of a thrombus, and growing along the course of the veins in the form of papillary masses hanging in the lumen. The growth passed through the heart into the pulmonary arteries, with metastatic deposits in the lungs. Similar deposits were found in the liver, brain, and intestine. The primary growth is designated by the author as lymphangioma testis.

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[1] Compt. rend. de la Soc. de Biol., April 6, 1900.

[2] Deut. Zeit. f. Chir., No. 50, 349, 1899.

[3] Contributions to the Sci. of Med., W. H. Welch's Festschrift, p. 497.

**The Occurrence of Eosinophile Leukocytes in Tumors.**—Feldbausch [1] details the results of an investigation of a number of tumors with reference to the occurrence within them of eosinophile leukocytes. In general his conclusion is that the occurrence of eosinophile leukocytes in a tumor is dependent upon the stage of the development of the growth; in the early stages they may be numerous, whereas with the growth of the tumor they become fewer, and may ultimately disappear. Distinguishing between glandular carcinomas and epitheliomas, he found that in the former the occurrence of these leukocytes was irregular, whereas in the latter they were never absent. The less degenerated a tumor, the more likely the occurrence of the leukocytes; the same is also true with regard to foci of necrosis, in the immediate vicinity of which they were seldom met. As regards the epitheliomas, it was only in the markedly degenerated tumors that the eosinophile leukocytes were absent. This is in accordance with the observations of Neisser [2] and Bettmann [3] regarding other affections of the skin—that is, in certain stages of the diseases there occurs an increase of the eosinophile leukocytes. In sarcomas the leukocytes were not of constant occurrence. In a case of lymphosarcoma with extensive metastasis, but without involvement of the bones, they were not encountered. This is interpreted as an indication that these cells are not formed within lymphoid tissue. Regarding the topographic arrangement of the eosinophile leukocytes, there was a marked regularity, in that they were encountered especially in the connective tissue that surrounded the tumor masses; this was particularly noticeable in epitheliomas. They were found, however, in the tumor tissue itself. As regards the origin of the eosinophile leukocytes, it is believed that they were derived from the blood; there was no ground for the supposition that they were formed *in loco.* Feldbausch suggests that the granules may bear a certain relationship to hemoglobin—that they may act as conveyers of oxygen to regions more or less inaccessible to the erythrocytes. Thus, when a tissue is invaded by bacteria or tumor masses, they immediately act to render the tissue more resistant. Or the granules may be looked upon as the active antitoxic material. In accordance with the recent suggestion of Buchner that the so-called alexins are to be sought in the leukocytes, these granules may be considered as the anatomic basis of the protective substances demonstrated by chemical means.

**The Pathologic Histology of Cyst-formations.**—Kühne [4] details the results of the examination of a series of cysts, which he classifies as follows: (1) Cyst-formations originating during extra-uterine life—(*a*) cysts of the small intestine, (*b*) cysts of the esophagus, (*c*) cysts of the spleen. (2) Cyst-formations originating during intra-uterine life. He prefaces his discussion of cysts of the small intestines by referring to the classification of Aschoff, who distinguishes 4 varieties of such cysts: (1) Cystic dilation of the chyle-vessels; (2) dilation of Lieberkühn's glands; (3) cystic dilation of the lymph-vessel network of inflammatory origin and containing serous fluid; and (4) cysts the result of closure of Meckel's diverticulum. He then reports 3 cases, in all of which there were distributed throughout the small intestine a number of sharply circumscribed cysts, of varying size, and containing a milky fluid. These are attributed to alterations in Lieberkühn's glands, on account of the epithelial lining, of the intimate association with the cysts of dilated glands of Lieberkühn, and of the absence of lymph-vessels. He believes that in consequence of inflammation (evident in the round-cell infiltration) the orifices of the glands became obstructed, distention progressed in consequence of the continuous accumulation of secretion, and ultimately the dilated glands became separated from the surface, either because of the round-cell infiltration or of proliferation of the mucosa. The partial degeneration and the differences in the height of the lining epithelium (in part high cylindric, in part low or cubic) are attributed to the effects of pressure. The elevations springing from the sides of some of the cysts are interpreted as an indication that the cysts resulted from the coalescing of a number of smaller ones—the elevations being the remnants of previous septums. Five cases of cyst-formations in the esophagus are also reported. Concerning these, it is stated that they occur only in those situations where glandular apparatus is found normally—that is, in the upper and lower thirds of the organ; that they are particularly prone to occur when, in consequence of inflammatory or other irritants, the orifices of the glands become occluded; that in the cyst-contents there may occur corpora versicolorata—bodies attributed to a combination of cell products and cyst-contents altered by stagnation or other causes; and that in those portions of the esophagus presenting epithelium resembling that of the stomach similar cysts may be encountered. "Gastric" mucous membrane was found in the upper part of the esophagus in one of the cases. This has also been observed by other authors, and has been attributed to fetal misplacements by Eberth. Schaffer, on the other hand, believes that under certain circumstances the entodermal cylindric epithelium, which gives rise to both the gastric epithelium and the esophageal epithelium, may lead to the formation in the esophagus of gastric glands. The latter opinion is concurred in by Kühne. He also reports 3 instances of cyst-formation in the spleen. The question whether these and similar cysts are lymph-cysts or the consequence of misplacement of peritoneal epithelium is considered. That two of the cases are the result of dilation of lymph-vessels is thought probable, because smooth muscle-fibers were found in the wall and lymphocytes in the contents. Smooth muscle-fibers have also been found by other authors in lymphangiomas and other lymph-cysts. It is considered likely that the third case is the result of proliferation of misplaced peritoneal epithelium (capsule epithelium). The report of two congenital cysts of the neck is prefaced by the following classification of Aschoff, who distinguishes these varieties of cysts: (1)

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[1] Virchow's Archiv, vol. CLXI, p. 1, 1900.
[2] Wien. med. Presse, Nos. 3, 4, and 5, 1892.
[3] Münch. med. Woch., No. 39, 1898.
[4] Virchow's Archiv, vol. CLVIII, p. 345, 1899.

Dermoid or branchiogenic cysts; (2) lymph-vessel cysts; (3) blood-cysts; (4) traumatic lymph-cysts; (5) foreign-body cysts; and (6) cyst-formations the consequence of inflammation of the mucous bursa situated in the median line of the neck. Of the two cases reported, the first, consisting of a wall made up largely of lymphoid tissue and lined with flat epithelium, is considered a typical branchiogenic cyst. The second, which was multilocular, was especially interesting on account of the presence in the wall of a large amount of muscle-fibers, lymphoid tissue, and glandular structure much resembling sweat-glands, and the lining consisting of cubic and cylindric epithelium. This cyst, which is thought to be unique, is attributed to disturbances in the development of the germinal ducts and the faucial arches. While that portion consisting of mucous membrane with glands is referred to the germinal ducts, the muscle-fibers are thought to have been derived from the pharynx, and a portion of the glands from the pharyngeal glands. Desiring to give this cystic formation a name, Kühne suggests "branchiogenic adenomyoma." In conclusion, he refers to von Kostanecki and von Milecki's classification of tumors provided with an epithelial lining and referred to proliferation of aberrant germinal-duct rests: (1) Tumors with simple epithelial lining. The epithelium may be (*a*) entodermal (cylindric or ciliated), (*b*) ectodermal (more or less resembling epidermis), and (*c*) entodermal and ectodermal side by side. (2) Rests of the germinal ducts with pathologically modified wall and correspondingly modified contents—(*a*) abscesses, (*b*) blood-cysts lined with epithelium, (*c*) adenomas and carcinomas, the consequence of atypical proliferation of the epithelium, (*d*) mixed tumors the result of combinations formed with tumors originating from the surrounding connective tissues. (3) Teratomas.

**The Histogenesis of Fibromyoma of the Uterus.**—Rizzuti [1] accepts the theory of Roessger, according to which the matrix of the new growth is formed by the walls of the small arteries.

According to Tridondani, [2] an examination of fibromyomatous nodules in their earliest stages permits one to exclude their formation from inflammatory elements or from embryonic germs. At the periphery of the nodules, between the myomatous and the uterine tissue, a zone of new connective tissue with processes of myomatous tissue and elastic fibers is found. In this, arterioles are scattered, the muscle-fibers of which are engaged in active proliferation. It is through the thickening of the arterial walls that, according to Tridondani, the myomatous nodules are formed. He assumes that there is a nerve stimulus produced by the menstrual hyperemia; in other words, that the stimulus for the proliferation originates in the ovary.

**The Histogenesis of Soft Nevi.**—Löwenbach, [3] after a critical summary of the various views promulgated regarding the histologic structure and pathogenesis of soft nevi, reports a personal observation: A man, aged 25 years, had had since birth or earliest childhood a nevus of the skin of the thorax. It was about the size of a hazelnut, had not increased in size for years, and was not especially pigmented. It was removed merely for microscopic examination, and was subjected to careful study in serial sections. Between the nevus-cells there could be detected bundles of tissue consisting of fibrils, newly formed elastic fibers, and connective-tissue nuclei. The epidermis, although there were collections of nevus-cells very near it, presented no alterations whatever—a finding at variance with the view of Unna. A relationship between columns of nevus-cells and blood-vessels was very evident; but whether the cells were within or about the vessels, they were everywhere separated from the wall of the blood-vessel—in the large vessel by distinct intima, media, and adventitia, and in the capillaries by endothelium and fibrous membrane. In many of the serial sections, however, it was evident that the nevus-cells resulted directly from proliferation of the endothelium of the vessels. The transition from endothelial cells to nevus-cells resulted in two ways: (1) Through an eccentric proliferation of the endothelium, in that the endothelium penetrated the connective-tissue membrane of the vessel and without the vessel gave rise to a column of nevus-cells that proliferated further without reference to the vessel; (2) through a concentric proliferation of the endothelium, whereby the lumen of the vessel was gradually constricted and finally obliterated by the production of a compact column of nevus-cells. This latter method was the more common, and is considered especially significant of the endothelial genesis of the specimen. Although believing that his specimen was of endothelial origin, the author states that all soft nevi may not arise in the same manner; some may be derived from the rete cells, and he cautions against generalizations from the observation of single cases.

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[1] Arch. Ital. di gin., No. 3, 1899; Centralbl. f. allg. Path. pathol. u. Anat., July 18, 1900.

[2] Ann. d'ost. e di gin., No. 5, 1899; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[3] Virchow's Archiv, vol. CLVII, p. 485, 1899.

**The Combination of Glioma and Tubercle.**—Kazowsky,[1] on examination of a tumor the size of a hazelnut attached to the right optic thalamus and right cerebral peduncle, found that it was composed of gliomatous and tuberculous tissue. He is of the opinion that the glioma was the primary condition.

**The New Formation of Elastic Tissue, Especially in the Stroma of Carcinoma.**—Williams [2] finds that when the stroma of the carcinoma is of new formation it is usually free from elastic fibers. The tumors in which newly formed elastic fibers occurred either contained a large amount of connective-tissue stroma, or the newly formed fibers were in connection with preexisting elastic elements of the original parts.

## MISCELLANEOUS.

**The Elastic Fibers in Giant Cells.**—Rona [3] has studied the behavior of elastic fibers in giant cells. The existence of such fibers was first pointed out by Ssudakewitsch. Some of the elastic fibers in giant cells are calcified; others by their staining show that they are not degenerated. Occasionally the fibers contain hemosiderin granules, as demonstrated by the Prussian-blue reaction. Ssudakewitsch was of the opinion that the presence of elastic fibers in giant cells is to be explained as a sort of phagocytosis—the cells surrounding the fibers and destroying them. Unna (and his view is shared by the author) believed that the giant cells grow around the elastic fibers, and that the degenerative changes in the elastic fibers are not brought about by an action of the giant cells, but are due to the same morbid influence that acts upon the cell itself. The point to be particularly explained is the long preservation of the elastic fibers in giant cells, since such fibers are very easily destroyed by inflammation. Thus, they are found in giant cells of tubercles, when very little elastic tissue, or none at all, is seen outside of the giant cells. The chemical process which brings about the destruction of the elastin in the tubercle must, in some way, have little influence upon the giant cells. This seems to indicate a slower metabolism in the latter.

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[1] Centralbl. f. allg. Path. u. pathol. Anat., May 23, 1900.

[2] Contributions to the Sci. of Med., W. H. Welch's Festschrift, p. 291.

[3] Beiträge zur pathol. Anat. u. zur allg. Path., Bd. XXVII, Heft 2, 1900.

**The Regeneration and New Formation of Elastic Tissue.**—It had been demonstrated by a number of investigators that regeneration of elastic tissue occurs in the intima of arteries, but the media had not been so carefully studied. This led Jores [1] to examine particularly the middle coat. He found that in thrombotic arteritis there was a decided new formation of elastic tissue in the media. After traumatic injuries produced in rabbits there was a complete regeneration of the elastic tissue in the media. No definite conclusions could be drawn concerning the adventitia. The investigation was also extended to connective-tissue tumors. Some fibromas are entirely devoid of elastic tissue; others contain greater or smaller quantities. Elastic fibers are constantly found in the fibromyomas of the uterus, in relation with the blood-vessels and also with the muscle-bundles. On the whole, however, connective-tissue tumors have but a slight tendency to the formation of elastic fibers. Elastic tissue is formed in interstitial inflammations, particularly in the liver and kidney; also in the myocardium and in cicatricial processes in the spleen, testicle, etc.

**Hemochromatosis: the Relation of Hemochromatosis to Bronzed Diabetes.**—E. L. Opie [2] reports a very interesting case of pigmentation of the skin associated with pigmentation of the liver, pancreas, heart, stomach, intestines, peritoneum, lymphatic glands, and testicles. Cirrhosis of the liver and chronic interstitial pancreatitis were also present. Death had been due to typhoid fever. Two kinds of pigment were found in the organs, one giving the iron reaction and the other an iron-free pigment. Hemochromatosis was first described by von Recklinghausen, and he characterized it as "the presence in the epithelial cells of various glands, notably of the liver and the pancreas, of an iron-containing pigment; the presence of an iron-free pigment in the smooth muscle-cells of the gastro-intestinal tract and of the blood-vessels and lymph-vessels, and in certain connective-tissue cells; the association of cirrhosis with pigmentation." French writers subsequently pointed out the existence of a form of diabetes associated with bronzing of the skin and cirrhosis of the liver. The characteristic lesions of the latter are pigmentation of the liver and pancreas, associated with cirrhosis, and the presence of an ochre-colored ferruginous pigment in the parenchymatous cells of the liver, pancreas, and other glands, and in the muscles of the heart, in the interstitial tissues of these organs, and in the lymphatic glands. The skin is usually, although not always, bronzed. Opie's case stands midway between von Recklinghausen's hemochromatosis and the so-called bronzed diabetes. It resembled the latter in everything except the presence of diabetes: that is, there was bronzing of the skin, cirrhosis of the liver of an advanced grade, and chronic interstitial pancreatitis. Regarding the relationship between the hemochromatosis and the diabetes, Opie believes that the latter is secondary to the former. His conclusions are as follows: (1) There exists a distinct morbid entity, hemochromatosis, characterized by the wide-spread deposition of an iron-containing pigment in certain cells and an associated formation of iron-free pigments in a variety of localities in which pigment is found in moderate amount under physiologic conditions. (2) With the pigment accumulation there is degeneration and death of the containing cells, and consequent interstitial inflammation, notably of the liver and pancreas, which become the seat of inflammatory changes accompanied by hypertrophy of the organ. (3) When chronic interstitial pancreatitis has reached a certain grade of intensity, diabetes ensues and is the terminal event in the disease.

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[1] Beiträge zur pathol. Anat. u. zur allg. Path., Bd. XXVII, Heft 3, 1900.

[2] Jour. of Exp. Med., May-July, 1899.

**The Origin of the Pigment in Addison's Disease.**—Pförringer,[1] in section of the skin from a case of Addison's disease, found the customary deposit of pigment in the basal portion of the epidermis; in the cutis the papillary layer contained numerous pigment-carrying cells, in part round and in part spindle-shaped or stellate. Pigment granules were also found free in the corium, usually in the neighborhood of capillaries. Granular pigment was present in the interior of capillaries, its color varying from yellowish-brown to black. In places a pigment mass could be seen to pass out between the endothelial cells, so that part of it was inside and part outside of the capillary. Some pigment was also found in the interior of leukocytes. The pigment did not give the iron reaction. These observations (similar ones have been made previously by von Kahlden) point strongly to the hematogenous origin of the pigment in Addison's disease. The pigment leaves the capillaries either free or in leukocytes, and is carried by connective-tissue cells to the epithelium. In view of the close approximation of the vessels to the epidermis, however, it is not improbable that there may be a direct transfer of pigment into the latter.

**Tissue and Metabolic Changes after the Extirpation of the Suprarenal Glands.**—The following are the conclusions reached by

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[1] Centralbl. f. allg. Path. u. pathol. Anat., Jan. 2, 1900.

Fabozzi[1]: (1) After bilateral extirpation life is possible up to 4 days; (2) when the glands are removed at separate periods, life is possible up to 25 days; (3) the female sex is the more resistant; (4) the suppression of the adrenal function brings about changes in metabolism; (5) it also causes marked changes in the liver, spleen, and cerebrospinal axis; (6) the adrenals exercise a tonic action on the vasomotor system; (7) the products retained after extirpation have a damaging influence on the nerve-centers; (8) the adrenals fulfil to a certain extent a protective function against infectious diseases.

**The Pathogenesis of Gout.**—Carboni and Generali[2] were unable to produce uratic deposits in the joint cartilages by the injection of solutions of urates into the bone-marrow, and even the introduction of neutral sodium urate or sodium biurate into the joints did not excite inflammatory or necrotic phenomena. Such phenomena were, however, readily produced by the intra-articular injection of adenin chlorid. This substance has also the property of forming with uric acid a compound which under certain conditions is insoluble. If these facts are brought into relation with the observation of Minkowsky, who produced uric acid infarcts by feeding dogs on adenin, the inference is justified that adenin plays an important rôle in the production of gout. It produces necrosis and inflammatory processes in the tissues, and at the same time unites with uric acid to form adenin urate. This is eventually transformed into sodium urate.

## TECHNIC.

**The Preservation of Stained Amyloid Organs.**—Davidson[3] recommends the following procedure for the preservation of amyloid organs: A section of the organ, as long and wide as is desired, but about 1 cm. thick, is washed in water to remove the greater quantity of blood from the vessels; it is then placed for a day in a weak solution of gentian-violet (10 drops of a 2% alcoholic solution to 150 cc. of distilled water). The surface of the section of the organ must be flat, and it must be completely immersed in the fluid, so that the particles of the stain that are precipitated fall upon the section. On the following day the preparation is violet; the amyloid areas, dark red. It is then washed in water and placed in a 1% solution of potassium acetate, to which should be added enough gentian-violet to render it pale blue (2 to 3 drops). On the following day the section should be again washed in water and placed in a 4% formalin solution, to which 2 or 3 drops of gentian-violet have also been added. In this solution the section may remain indefinitely. In the course of time the colors change somewhat, in that the violet areas become pale and grayish, whereas the reddish amyloid areas become somewhat bluish. But the contrast is still very marked, and is especially evident if the surface of the section be viewed through a piece of glass pressed against it.

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[1] Gior. internaz. della Scienze mediche, 1899, IV; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[2] R. Accad. di Torino, July 13, 1899; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[3] Virchow's Archiv, vol. CLIX, p. 570.

**A New Neuroglia Stain and the Nature of the Glia Fibers.**—Yamagiwa [1] publishes the following staining procedure: Exceedingly thin sections of the brain or spinal cord are first hardened in Müller's fluid, the fluid being renewed every 5 or 6 days. From the Müller's fluid, the sections are placed directly, without washing, in absolute alcohol, wherein they remain several days to a week, the alcohol being renewed every day. They are then embedded in celloidin and cut in the usual manner. The following are the stages of the staining method: (1) Place the sections in a saturated or concentrated alcoholic solution of eosin 12 hours or longer; (2) in a concentrated watery solution of anilin-blue 4 to 6 hours; (3) differentiate by immersion in a weak alcohol rendered slightly alkaline by the addition of 1 % potassium hydrate; the deep-blue color of the section changes instantly or gradually, depending upon the alkalinity of the alcohol, to a reddish-brown color; (4) remove the alkaline alcohol by washing in distilled water; (5) remove the excess of anilin-blue in dilute alcohol; the sections reveal a reddish color; (6) dehydrate in absolute alcohol; (7) clear in oil of origanum, wherein the sections become somewhat more blue; (8) mount in balsam. The axis-cylinders are deep blue; the glia fibers and the red blood-corpuscles, dark red; the medullary sheaths, bright red; the protoplasm of the glia cells, pale violet (or bluish-red); the cell-body of the ganglion cells, pale bluish-green (with greenish granules); their thick processes, pale bluish; connective-tissue fibers, adventitia, and intima of the blood-vessels, sky-blue to pale greenish; the media, bluish-red; all nuclear membranes, bluish; the nucleoli of the ganglia cells, deep violet or deep blue; and the nucleoli of the glia cells, also bluish, but with a shade of red. As regards the nature of the glia fibers, he believes that his stain demonstrates that they are the differentiated peripheral portion of the glia cells, the cell-bodies of which are not entirely or sharply circumscribed by a special membrane.

**A New Method for the Study of the Nerve-cell Processes.**—Cominelli [2] recommends the following method: Fixation in Müller's fluid or in alcohol; embedding of thin sections in paraffin. The paraffin is removed from the cut sections and the latter are washed in absolute alcohol and kept for 24 hours in ordinary alcohol. Thence they are transferred for 3 minutes to a watery alcoholic solution of hematoxylin prepared by adding as many drops of the alcoholic solution as there are cubic centimeters of water. The sections are then washed in water and placed for 5 minutes in a 1 % solution of potassium permanganate. After washing in water, this is repeated 3 or 4 times. The sections are dehydrated with alcohol of increasing strength and placed in oil of cloves and Canada balsam. The nerve elements take on a greenish-black color and are thus very suitable for photography.

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[1] Virchow's Archiv, vol. CLX, p. 358.

[2] Il Policlinico, No. 11, 1899; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

**A Modification of Nissl's Method of Staining the Nerve.**—Boccardi [1] hardens the tissues in absolute alcohol or in formalin with picric-sulphuric acid. The staining fluid consists of erythrosin, 10 cg.; toluidin-blue, 20 cg. to 25 cg.; water, 100 gm. Sections are stained from 20 to 25 minutes and washed in water, differentiated in a 0.5% alum solution, washed in water, then in 70% alcohol, placed in absolute alcohol, and then in xylol and balsam.

**Mixtures of Eosin and Methylene-blue as Staining Agents.**—When solutions of eosin and methylene-blue are mixed, a neutral dye is produced which has valuable staining properties. To obtain a proper mixture the dyes are dissolved in proportion to their molecular weights. Of a 1% solution of eosin 1000 cc. are mixed with 882 cc. of a solution of 1% of methylene-blue. A neutral dye is precipitated in about 48 hours. Laurent [2] has had a solution of the precipitate prepared by Grubler. One part of the solution is added to 4 parts of water and boiled in a test-tube. After quickly cooling the solution, the object to be stained is immersed in it for from 1½ to 6 hours. If the preparation is a cover-glass, it is dried between filter-paper and moved backward and forward in absolute alcohol until clouds of color are no longer given off. It is then placed in pure xylol and embedded in thickened cedar oil. After being removed from the stain, sections are momentarily washed in 90% alcohol, and then placed in absolute alcohol, where they may remain as long as 6 hours. Ordinarily, from 2 to 10 minutes suffice. Instead of absolute alcohol, anilin-oil xylol or anilin-oil alcohol can be employed. Afterward the sections are placed in xylol and embedded.

**Eosinate of Methylene-blue as a Blood Stain.**—Simon [3] recommends eosinate of methylene-blue for purposes of staining, as well as fixing blood films. The stain is made by adding an equal part of a 1.2% watery solution of eosin to a 1% solution of methylene-blue in an open basin, which is allowed to stand for 24 hours. The precipitate of eosinate of methylene-blue is filtered off and dried at ordinary temperatures. The powder is then washed with distilled water, again dried, and stored in suitable receptacles. The crystals are insoluble in cold water, very slightly soluble in hot water, and are readily dissolved in alcohol or in solutions of eosin or methylene-blue. For staining purposes Simon employs a saturated solution of the dye in alcohol, to which 10% of glycerin is added. The blood is spread as usual upon slides. When they are dried, they are covered with a few drops of the solution, which is allowed to remain from 2 to 5 minutes, after which they are rinsed in water and thoroughly dried by slow evaporation and subsequent mild heating. The individual granules in the neutrophilic leukocytes are stained a purple. Polychromatophilic and granular degenerations of the red corpuscles are readily recognized. With diabetic blood a moderate

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[1] Riforma med., 1899, LV, 69; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[2] Centralbl. f. allg. Path. u. pathol. Anat., Feb. 15, 1900.

[3] Maryland Med. Jour., April, 1900.

Bremer's reaction is obtained. The red corpuscles are colored a pale-green or yellowish-green, or they remain uncolored. The basophilic leukocytes present a violet granulation, and malarial organisms as well as bacteria are stained a sky-blue. He regards this stain as a decided advance in the technic of blood examination.

## THE BLOOD.

**Leukemia as a Protozoan Infection.**—Löwit,[1] by means of special staining methods, has discovered in the blood of leukemia peculiar bodies which he interprets as protozoa. The blood films are fixed for from 1 to 2 hours at a temperature of from 110° to 120° F., not in alcohol and ether, and then are placed for half an hour at room-temperature in a concentrated watery solution of thionin; are washed, dried in the air, and laid for from 10 to 20 seconds in a watery solution of iodin and iodid of potash (iodin, 1; potassium iodid, 2; distilled water, 300). After drying they are embedded in balsam. The parasites appear of various shades of green; the basophilic granules and the karyolytic and cytolytic products, dark bluish-red or brownish-red; the protoplasm of the leukocytes and the other specific granulations, yellowish and yellowish-brown; the leukocyte nuclei, pale-brown and sometimes reddish-gray; the erythrocytes, yellow; and the nuclei of erythroblasts, dark-brown. Various other modifications of staining were tried, but it is not necessary to detail these. In the blood of myelemic cases Löwit found peculiar bodies with metachromatic staining, lying in or against the leukocytes, especially the lymphocytes, and never in the red corpuscles. The larger ones showed segmentation phenomena, with sickle-, spindle-, or crescent-shaped formations. Löwit believes that these bodies are different from the basophilic granules, and that they can be readily distinguished from blood-plates, fat, glycogen, amyloid, etc. Control examinations with human and animal blood were negative. In unstained fresh blood Löwit was able to find appearances that might be interpreted as specific bodies in only one case. They were feebly motile. The small segments of the parasite are looked upon as juvenile forms—as sporozoites. No membrane was visible about the parasite. In methylene-blue staining a depression in the ectoplasm and endoplasm was at times visible, and the existence of flagellæ was inferred from the quick, jerky movements of blood-corpuscles in the fresh preparation. After much labor Löwit succeeded also in demonstrating the organisms in sections of tissue. The parasites belong to the group of Leukocytozoa of Danilewsky, and are divisible into 3 forms: juvenile forms—small ameboid discs or sickles; large amebic or falciform bodies; and morular forms, resembling the sporulation stage. The blood-forming organs, particularly the spleen, are richer in parasites than the blood, and may be considered as the breeding-grounds. To distinguish the parasite from a smaller form found in lymphemia, Löwit designates it as Hæmamœba leukæmiæ magna. In 5 cases of lymphemia smaller bodies were found, which are designated Hæmamœba lymphæmiæ parva (vivax). Experiments were made, principally on rabbits. The typical picture of leukemia was not obtained, but a disease resembling it; and Löwit does not, therefore, speak of it as leukemia of rabbits, but as "leukemic infection" in rabbits. The transmission has so far succeeded only in myelemia, and it is a necessary condition for success that the material be thoroughly triturated, so that the ameba may become free. The ameba could be demonstrated in the blood, even in the form of flagellate bodies. Artificial cultivation did not succeed. Regarding the pathology of leukemia, Löwit does not accept the views of Walz and Neumann as to the myelogenic origin of most forms of leukemia, but maintains that the primary seat is in the blood. He believes that the parasite infects the blood and primarily destroys the juvenile forms, producing a constant accession of new lymphocytes, which accounts for the hyperplastic processes of the blood-forming organs. If, now, the parasite locates in these organs themselves, a new irritation is produced, and the regenerative and degenerative processes are intensified. The deciding factor regarding the form of leukemia is the parasite—not the changes in the bone-marrow; hence the designation of the form of leukemia is not to be based on the hematopoietic organs, but on the differences in the leukocytes, and Löwit proposes to speak of poikilocyte and polymorphocyte leukemia, produced by Hæmamœbæ leukæmiæ magna, and homoiocyte leukemia, corresponding to the lymphemia or lymphocyte leukemia of Walz, produced by the Hæmamœba leukæmiæ parva. [Walz was able to demonstrate the peculiar bodies in a case of myelemia, but is not prepared to interpret them as parasites. With the special staining so clearly described by Löwit, it should not be difficult to repeat his investigations.]

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[1] Die Leukämie als Protozoeninfektion, Untersuchungen zur Aetiologie und Pathologie, Wiesbaden, 1900; Centralbl. f. Bakt., Parasit. u. Infektionskrankh., Mar. 31, 1900.

**Leukemia.**—Löwit [1] in two additional cases of leukemia describes the presence in the lymphocytes of peculiar ring-shaped bodies of about one-sixth to one-third the size of a red corpuscle. In the bodies of leukemically infected rabbits he found flagellated forms. In human beings the hemamebas multiply by simple sporulation; in animals, in which the conditions are less favorable, a sexual propagation occurs analogous to the reproduction of malarial plasmodia.

In 3 cases of myelemia (polymorphocyte leukemia) Löwit [2] was able to demonstrate the presence of Hæmamœba leukæmiæ magna, and, in addition, he claims to have observed appearances suggesting a sexual propagation. In 2 cases of lymphemia (homoiocyte leukemia) he found within the circulating leukocytes parasite-like inclusions, which bore a relation to the nucleus, and were different from the parasite of myelemia. The author proposes to call them, instead of Hæmamœba parva (vivax), Hæmamœba leukæmiæ parva intranuclearis. The parasite was also found in the blood-forming organs.

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[1] Eighteenth Cong. for Intern. Med., April 18–21, 1900; Centralbl. f. allg. Path. u. pathol. Anat., May 23, 1900.

[2] Centralbl. f. Bakt., Parasit. u. Infektionskrankh., April 20, 1900.

Türk [1] is of the opinion that the hemameba described by Löwit is nothing more than a mast-cell that has lost its granulation by reason of the stains used. The acid solution of basic dyes brings about a disappearance of the granulation, so that the resulting cell looks like the hemameba.

**Friedländer's Bacillus in a Case of Acute Leukemia.**—Mariotti Bianchi [2] obtained Friedländer's bacillus in pure culture from the pleural exudate, the spleen, and the bone-marrow, in a case of acute leukemia, and concludes, on the basis of this case, and those reported in the literature, that acute leukemia is an infectious disease which can be brought about by a variety of bacteria, all having a tendency to produce hemorrhagic lesions.

**Observations Concerning Leukemic Lesions of the Skin.**—In Oertel's [3] case small cutaneous nodules of irregular distribution developed, which were thought to be multiple sarcomas. An accurate examination of the blood was not made, but an unstained specimen showed an enormous increase in the leukocytes, many of which were of the type of myelocyte and many were eosinophiles. The patient died and an autopsy hedged about by restrictions was made, and it was difficult to obtain material for histologic study. The spleen was much enlarged and presented old infarcts. Chronic parenchymatous nephritis of the hemorrhagic type, chronic adhesive peritonitis, and fatty liver were also found. Examination of the cutaneous nodules showed them to be leukemic deposits. The epidermis and the upper part of the cutis showed no definite changes. The lower part of the cutis, however, was the seat of a dense and diffuse small-celled infiltration. This infiltration extended upward, penetrating the fibers of the cutis, either replacing them entirely or separating them into bundles. Only a very fine reticulum of connective-tissue fibrils remained, which contained groups of cells. The glands were embedded in the cellular masses; they did not show a direct relation to the process. The earliest changes were represented by small, circumscribed focal accumulations of cells situated in the lower part of the cutis, visible to the naked eye. As the process progressed these small nodules became confluent, and assumed the character of a diffuse infiltration. The cells had the appearance of leukocytes, the largest part of which were lymphocytes; of these, 2 kinds could be distinguished: (1) small cells with narrow, faintly staining cytoplasm and deeply staining nucleus, and (2) large cells with feebly staining cytoplasm and a large, somewhat indented and irregularly staining nucleus. Polymorphonuclear leukocytes were common as well as eosinophilic cells with bright red, coarse granulations. There were very few red corpuscles, and plasma cells were seen in very small number and formed no characteristic part of the cellular accumulations. The nodules were evidently composed of cells derived from the blood.

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[1] Eighteenth Cong. for Intern. Med., April 18-21, 1900; Centralbl. f. allg. Path. u. pathol. Anat., May 23, 1900.

[2] Riforma med., 1899, II, 6; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[3] Jour. of Exp. Med., 1899, p. 569.

**The Rôle of Iron in the Formation of Blood and the Nature of Chlorosis.**—Hofmann [1] has detailed the results of a long series of investigations with reference to the rôle of iron in the formation of blood. The investigations, which were made upon 98 rabbits, comprise the histologic examination of organs ordinarily looked upon as hematogenic; the enumeration of the blood-corpuscles; the estimation of the hemoglobin; the study of the pathways whereby the iron reaches the organs; the utility of the different preparations of iron; the action of the latter in healthy and in anemic animals, etc. He concludes that the iron, no matter in what form it is administered, is absorbed from the duodenum. Being taken up by, and combining as an albuminate with, the leukocytes, which act as carriers, it circulates in the blood. In this form the iron has no toxic action. It can be found in abundant quantity, not only in the ordinary storehouses,—the spleen and the liver,—but also and especially in the bone-marrow. Here, in consequence of the delayed circulation of the blood, iron-laden cells are present in large number, not only in the parenchyma of the marrow, but also in the wide blood channels that form a network between them. After loss of blood the bone-marrow only presents a corresponding regenerative activity—rendered evident by a very marked hyperplasia of its parenchyma. The replacement of the red blood-corpuscles is more rapid and the marrow is richer in all its constituents in animals to which iron has been given than in those that have received none; the spleen and lymph-glands, however, present no differences. In addition, in animals in which there has been no loss of blood the exhibition of iron results in a moderate increase in the number of blood-corpuscles in the circulating blood. The bone-marrow, however, presents no marked proliferation of cells, but it does reveal an increase of fat. The replacement of the hemoglobin is less rapid than that of the corpuscles; but whether or not iron has been administered, it occurs *pari passu* with that of the corpuscles—that is, an independent increased production of hemoglobin does not result from the administration of iron. From this it follows that the action of the iron, as such, is to stimulate the physiologic activity of the marrow—to hasten the ripening of the young forms of blood-corpuscles produced in the marrow and to expedite their entrance as nonnucleated corpuscles into the circulating blood. From investigations undertaken with various so-called organic preparations of iron, and with preparations of hemoglobin, it was determined that the effect of iron therapy depends not upon the preparation of iron, but upon the quantity of the metal absorbed. From this specific action of the iron to stimulate the physiologic activity of the bone-marrow one may gain an insight into the nature of chlorosis, in which disease the administration of iron is attended by unerring success. This disease in all probability consists either in a transitory diminished functional capacity of the blood-forming organs (the bone-marrow), developing only at puberty, or in a congenital hypoplasia of the marrow that renders itself more or less evident throughout the life of the individual. In severe cases these are associated with the hypoplasia of the vascular apparatus, to which Virchow directed attention; also at times with hypoplasia of the sexual organs. This weakness of the blood-forming organs manifests itself in the production of diseased erythrocytes—erythrocytes defective in form and hemoglobin. While all other theories concerning the nature of chlorosis are inconsistent with the specific action of iron, that here given finds support in the results of venesection, which is likewise a stimulant to the blood-forming bone-marrow. [This theory of chlorosis is by no means new, and has been maintained by Von Noorden and others.]

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[1] Virchow's Archiv, vol. CLX, p. 235.

## THE CIRCULATORY SYSTEM.

**A Case of Acute Endocarditis Caused by Micrococcus Zymogenes.**—MacCallum and Hastings[1] report the case of a man who was supposed to be suffering from a relapse of typhoid fever. The examination of the heart showed decreased area of dullness accompanied by a pure diastolic murmur heard over the body and base of the heart, suggesting disease of the aortic valves. The pulse also indicated aortic incompetency. The urine contained albumin and gave the diazo reaction. The temperature was high, there was a leukocytosis of 18,000, and a Widal reaction was present. The patient rapidly became worse, and endocardial murmurs and a thrill that suggested mitral as well as aortic disease developed. At this time agar plate cultures were made that resulted within 48 hours in the growth of what appeared to be a short-chained streptococcus, which subsequently proved to be a micrococcus growing for the most part in pairs. Three days before the patient's death blood cultures yielded the same organism. The patient died of cardiac failure, and at the autopsy the principal lesion was subacute and acute ulcerative endocarditis of the aortic and mitral valves. There were also an acute splenic tumor, septic infarctions in the spleen and kidneys, embolic abscess in the intestinal wall, bronchopneumonia, and chronic diffuse nephritis. Agar cultures were made from the heart's blood, valvular vegetations, gall-bladder, and splenic and renal infarctions, in all of which numerous pin-point, somewhat opaque, white colonies developed. These colonies gave a single organism that was identical with the organism cultivated during life. It is an extremely minute micrococcus, often somewhat elongated or elliptic in outline. It is usually found in pairs, is not motile, stains with the ordinary stains, and remains deeply stained by Gram's method. It grows on the ordinary mediums, does not produce agar nor indol, and liquefies gelatin slowly. The most characteristic features are seen in milk, which it acidifies, coagulates, and subsequently liquefies. It produces a milk-curdling ferment and a proteolytic ferment, each of which is separable from the bacterial cells. In old cultures it remains viable for months and is tolerably resistant to the action of heat and antiseptics. It is pathogenic for mice and rabbits, causing either abscess or general infections. Typical acute vegetative endocarditis was experimentally produced by intravenous inoculation in a rabbit and a dog, and the cocci were demonstrated in pure culture in the vegetations and in other parts of these animals after death. The organism is named Micrococcus zymogenes.

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[1] Jour. of Exp. Med., 1899, p. 521.

**The Relation of Myocarditis to Disease of the Coronary Arteries.**—Fujinami,[1] having investigated carefully 52 hearts with reference to the relation between myocarditis and disease of the coronary arteries, comes to the following conclusions: (1) The acute, circumscribed, parenchymatous myocarditic foci correspond in situation always with a point of marked narrowing or complete closure of the nutrient artery. (2) Fibrous myocarditis and arteriosclerosis are generally combined. The situation and the degree of the arteriosclerosis, however, vary. Most frequently there is a marked arteriosclerosis in the course of the coronary artery. There may, however, occur a slight arteriosclerosis throughout the course of the artery in association with well-marked fibrous myocarditis. The arteriosclerosis may even be circumscribed to the root of the aorta. Special emphasis is to be directed to the fact that in none of the cases examined did the coronary arteries and the root of the aorta exhibit the same degree of arteriosclerosis. (3) In the majority of the cases a marked constriction of the lumen of the coronary artery was not found regularly in the center or at the periphery of the individual fibrous foci. On the contrary, the areas markedly constricted by sclerosis were found at almost any situation along the small coronary branches. (4) In but a relatively small number of the cases could the narrowing or complete obstruction of the lumen of the coronary artery be demonstrated as the direct cause of the fibrous myocarditis, in that it was in direct association with the fibrous focus. (5) Indirectly, however, the narrowing of the lumen of the artery may give rise to the fibrous myocarditis. Although the constriction may be independent of—that is, removed from—the fibrous focus, still it may give rise to such marked disturbance of the circulation as to lead to degeneration of the muscle-fibers and a consequent reactive inflammation. These marked alterations of the vessel-wall may be situated either in the large coronary branches, or entirely without the heart, in the root of the aorta, or at the point of origin of the coronary arteries. (6) In addition to the fibrous myocarditis with previous muscle-fiber degeneration, there occur cases of primary, interstitial, nonsuppurative myocarditis. (7) If the muscle-fiber degeneration is but slight in extent, it is not of necessity the result of arterial obstruction; it is more likely the consequence of the action of a toxin. Later, however, the focus develops into a typical, fibrous, myocarditic area. (8) The mode of origin of fibrous myocarditis, therefore, is not always the same. This anatomic condition, on the contrary, is to be looked upon as the end-result of different processes. (9) Thickenings of the blood-vessels demonstrable microscopically are not always to be looked upon as the real cause of the fibrous foci; they may merely accompany the latter or be their cause, and they may bear the same relation to the myocarditis as the blood-vessel alterations found in other chronic processes, in cicatrices, and in connective proliferations of tumors due to these conditions. (10) Arteriosclerosis of the coronary arteries, therefore, is frequently the etiologic factor in the production of myocarditis; in many cases of fibrous myocarditis, however, it bears only a coordinated relationship to this condition, and can be viewed as a complication only. (11) Partial aneurysm of the heart occurs as a consequence of sclerosis of the coronary arteries. (12) The arteriosclerosis may also be the indirect cause of rupture of the heart. (13) The combination of fragmentation of the myocardium and sclerosis of the coronary arteries and fibrous myocarditis is remarkably common. This combination is probably not accidental.

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[1] Virchow's Archiv, vol. CLIX, p. 447.

**Tumors of the Heart.**—Geipel[1] reports 2 interesting cases. One was a round-cell sarcoma of the right auricle, apparently primary, in a woman of 53. There had been a metastasis to the right ventricle. The tumor was accidentally discovered, having seemingly not caused any symptoms during life. In the other case, a man of 70, the tumor was secondary to a cylindric epithelioma of the bronchus. The tumor had grown into one of the pulmonary veins, and thence into the left auricle, three-fourths of the cavity of which it filled. Primary tumors of the heart are most frequent in the auricles. The right ventricle is very rarely involved. Secondary tumors are not rare in the heart. Probably carcinomas of the esophagus are more commonly the cause of cardiac involvement than tumors elsewhere.

**Primary Fibromyxoma of the Left Auricle of the Heart.**—Jacobsthal[2] reports the case of a child, aged 4 years, which came to necropsy with the clinical diagnosis of mitral insufficiency. The necropsy, however, revealed a tumor of the left auricle, which had led to hypertrophy of the heart, especially of the right ventricle. There was no thickening or other alteration of the mitral valves or endocardium in the neighborhood of the tumor. The latter was 6 × 3 × 3 cm., of grayish-red color, and of colloid consistence. Microscopic examination revealed it to be a fibromyxoma originating from the connective tissue of the endocardium. The tumor was especially interesting on account of the great amount of elastic fibers it contained.

**Endophlebitis.**—A. V. Meigs[3] very carefully describes the histologic features of endophlebitis, the studies having been made on the thickened vein on the dorsum of the foot of a man of 25. The intima was enormously thickened and was thrown into papillary folds, which almost occluded the lumen. In many respects the histologic picture resembled that seen in syphilitic endophlebitis. There was, moreover, a history of syphilis in the patient. Meigs is, however, of the opinion that other conditions may produce endophlebitis of the character described. [The picture of syphilitic endophlebitis of the mesenteric veins in the article of Forssman [1] is almost identical with that accompanying the paper of Meigs.]

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[1] Centralbl. f. allg. Path. u. pathol. Anat., Nov. 20, 1899.
[2] Virchow's Archiv, vol. CLIX, p. 351, 1900.
[3] Proc. Path. Soc. of Phila., Nov. 1, 1899.

**The Anatomy of Varicose Veins of the Lower Extremity.**—It has hitherto been the general opinion that the saccular dilations so frequently seen in the varicose saphenous vein occur just above valves, but Slawinski [2] has shown that these saccular dilations always occur just below the valves. At times the upper portion of the diverticulum is situated at the junction of the valve and vein; usually it is situated about 1 cm. below the valve.

## THE URINARY ORGANS.

**The Alterations of the Small Arteries in Diseases of the Kidneys.**—Friedemann,[3] in a study of the alterations of the small arteries in diseases of the kidneys, utilized not only the ordinary methods of histologic investigation, but also the Unna-Tanzer and Weigert elastin stain. He concludes that in all forms of chronic interstitial nephritis there occur alterations of the small arteries of the body which consist essentially of hypertrophy of all three coats of the vessels. The thickening of the intima heretofore described as fibrous endarteritis does not consist of ordinary fibrillar connective tissue, but of newly formed elastica. However, the smaller arteries often exhibit arteriosclerosis, which may be distinguished from hypertrophy of the artery by the fact that in the former the thickening of the media does not consist of increase of the muscle-fibers, but of increase of the connective tissue. In the intima there is found to the inner side of the newly formed elastic membrane a fibrous or hyaline layer, in which evidences of degeneration of the contained elastic tissue may always be observed, at times also vascularization and foci of necrosis. Both alterations are often to be noted in the same vessel, so that a sharp distinction is not always possible. In a large number of the cases, however, the hypertrophy of the vessel-wall occurs alone. The cause of this is the augmented blood pressure in interstitial nephritis, and the increased resistance producing the augmented blood pressure is to be found in the capillary system. The newly formed elastic membranes result from fibrillation of the elastica interna and resultant proliferation of the cells normally found between the lamellæ of the elastic membrane. In addition, it is probable that the formation of elastic fibrils forming themselves into membranes occurs also in the media.

**The Nature of Toxic Nephritis.**—Lindermann [4] has found that the introduction of finely divided renal substance from one species of animal into an animal of another species endowed the serum of the latter with a *nephrolytic* property; that is to say, the serum acquired the power of acting as a poison on the kidney of the animal species the renal substance of which had been employed. He also discovered that the serum of animals poisoned with chromate of potash was capable of producing nephritis in other animals. His conclusions are: (1) That the serum of dogs poisoned with potassium chromate when injected intravenously into healthy dogs acts as a renal poison, producing marked albuminuria, and may even lead to uremia. (2) The symptoms of lethal poisoning are marked depression of the temperature, reduction in the elimination of the normal urinary constituents, and, finally, fatal uremia. (3) The symptoms of a nonfatal intoxication are diminution in the quantity of urine and albuminuria, lasting from 3 to 5 days. (4) At autopsy the kidneys are enlarged and hyperemic; or, after a more chronic course, pale and mottled with hemorrhages. Microscopically, an acute, hemorrhagic nephritis is found. (5) The toxic properties of the blood-serum are not due to the retention of chromium compounds, as these are very quickly eliminated from the organs. They are found only in the liver and kidney; not a trace can be discovered in the blood. (6) After recovery from poisoning a transitory immunity exists. (7) Other renal poisons seem to have the property of rendering the serum of animals toxic. (8) The serum of animals of other species also acquires toxic properties when chromic acid is introduced. (9) The normal dog serum, even in large doses, does not produce albuminuria in dogs.

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[1] See p. 346 of this volume.

[2] Centralbl. f. allg. Path. u. pathol. Anat., Dec. 30, 1899.

[3] Virchow's Archiv, vol. CLIX, p. 541.

[4] Centralbl. f. allg. Path. u. pathol. Anat., May 23, 1900.

**Chronic Interstitial Nephritis and Arteritis in the Young and Family Nephritis; Calcification in the Liver.**—Brill and Libman [1] report the case of a girl, aged 14 years, who came from a family in which chronic interstitial nephritis was common, the 3 eldest children presenting the disease, one of whom was the patient. The patient had symptoms of chronic interstitial nephritis and general arteritis, and died as a result of the disease. At autopsy the kidneys were found to be among the smallest on record in the chronic nephritis of early life; the right kidney weighed 59 gm. and the left 34.5 gm. Microscopically, the changes incident to chronic interstitial nephritis were noted. The liver contained areas of calcification that looked as though they began with changes in the walls of the arteries and that the parts supplied by the arteries then became degenerated and infiltrated with lime-salts. The arterial change was very extensive and quite marked; the heart was markedly hypertrophied; the aortic valves were atheromatous; the coronary arteries, the aorta, the endocardium, the arteries in the organs, etc., showed marked atheromatous change. The disease induced a hemorrhagic diathesis, which was manifested by cerebral hemorrhage and by hemorrhages into the lung, spleen, and mesentery.

A case of **osteoid chondrosarcoma of the bladder** in a man aged 72 years is reported by Beneke,[2] who remarks the rarity of the tumor, and discusses at length the question of metaplasia and anaplasia. The tumor, the duration of which was indefinite, was about the size of a small apple; its surface was cauliflower-like, but it required a saw to section it. Microscopically it was composed of sarcomatous, cartilaginous, and osteoid tissue. The question of the metaplasia of these structures is discussed at length. It is deemed most probable that this and several other cartilaginous tumors of the bladder result from proliferation of aberrant rests.

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[1] Jour. of Exp. Med., 1899, p. 541.

[2] Virchow's Archiv, vol. CLXI, p. 70.

**Primary Sarcoma of the Prostate in a Boy 3¼ Years Old.**—Schalek [1] reports a case of primary sarcoma of the prostate in a boy 3¼ years old. The tumor was a spindle-cell sarcoma with mucoid and hemorrhagic change. Prostatic glandular tissue was found in its middle portions. Regarding the etiology, the author expresses the rather unsatisfactory opinion that the predominance of the fibromuscular tissue in the prostate over the glandular portion in childhood is the causal factor.

**Urinary Toxicity and Albuminuria.**—From a study of 120 specimens of urine Labadie-Lagrave, E. Boix, and J. Woé [2] conclude: (1) That there is no relation between the presence or the quantity of albumin found in a urine and the coefficient of toxicity of that urine; (2) that the gravity of the prognosis of a case of Bright's disease or the actual condition of the patient should be judged not upon the presence, the absence, or the quantity of albumin, but upon the coefficient of urinary toxicity.

**Bence Jones' Body.**—According to Magnus-Levy,[3] this body is found most frequently in the urine in myeloma, but in a case of leukemia and one of myxedema it was also present. It is precipitated by heating, but is usually redissolved at 100° C. Digestion with pepsin gives protoalbumoses and deuteroalbumoses. The body is therefore an albumin, and not an albumose. The author claims to have obtained it in crystalline form, and is inclined to consider it a normal product of the urine. In the discussion Senator stated that so far it had been found only in bone disease, but Von Jaksch was not able to obtain it in one case of extensive bone disease.

**Ethereal Sulphates and Indican in the Urine, and Their Relation to Each Other.**—There is, according to Scotti,[4] no constant relation between the quantity of ethereal sulphates and indican in the urine. Both may be present in considerable quantities without any marked disturbance of the digestive tract.

**Alimentary Oxaluria.**—Pierallini,[5] as the result of the experimental administration of oxalic acid and calcium oxalate, and the estimation by the Salkowski method of the quantity of oxalic acid excreted in the urine, concludes that the soluble and insoluble salts of oxalic acid are in part absorbed, the latter in smaller quantity, the former in larger quantity; that they are to be found in the urine as calcium oxalate; and that the quantity of these salts contained in the ordinary articles of diet is sufficient to cause an increase in their excretion.

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[1] Prag. med. Woch., Nos. 43 and 44, 1899.

[2] Comp. rend. de la Soc. de Biol., Feb. 23, 1900.

[3] Eighteenth Cong. for Intern. Med.; Centralbl. f. allg. Path. u. pathol. Anat., May 23, 1900.

[4] La nuova Rev. clin. terap., 1899, VIII; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[5] Virchow's Archiv, vol. CLX, p. 173, 1900.

## THE DIGESTIVE TRACT.

**The Toxicity of the Human Saliva.**—Pignatti Morano and Baccarani [1] tested the toxicity of saliva from healthy and diseased human beings on rabbits. They found that the saliva was toxic in doses of 20.738 cc. per kilo of animal. The toxicity varied with individuals within wide limits, and seemed to have very little relation to health or disease. There was also no relation to be found between the toxicity, the specific gravity, the degree of alkalinity, or the quantity of ptyalin or mucin. The animals generally died in convulsions.

**Experimental Reproduction of Dental Caries.**—After having made a small cavity in the first right lateral incisor of a sheep J. Choquet [2] placed a small quantity of a gelatin culture of a micro-organism obtained from human teeth filled for from 4 to 7 years in the cavity, using all precautions. The cavity was then closed by cement and the cement covered with hot wax so as to prevent the penetration of saliva. The animal was killed 9 months later, and after removing the filling from the cavity the dentin was found slightly yellow in color and was somewhat softened. A portion of this soft dentin inoculated into a suitable medium gave rise to a growth of the micro-organism used to make the experiment. This organism is a facultative anaerobic bacillus, which grows on gelatin and in bouillon. Its medium of predilection is one composed of 500 gm. of bouillon, 36 gm. of gelatin, 5 gm. of peptone, and 5 gm. of glycerophosphate of lime. It is motile in the hanging drop; stains with the usual dyes, but will not take Gram's stain.

**Sarcoma of the Esophagus.**—Gastpar,[3] after reviewing briefly the literature of the subject, reports a case of sarcoma of the esophagus in a man of 54. The tumor, which did not occlude the esophagus completely, was 14 cm. long, extended down into the stomach, and had the appearance of firm, smooth cauliflower growths, some of which depended into the stomach. Microscopically the tumor revealed itself as a large-celled polymorphic sarcoma. At autopsy it is possible to make a differential diagnosis of sarcoma of the esophagus from carcinoma on finding that the tumor is smooth, firm, and coarsely lobulated, and has very little ulceration.

**Pathology of Gastrosuccorrhea.**—Albu and Koch [4] report the histologic findings in a case of gastrosuccorrhea. The case had been one of chronic hydrocyanic acid poisoning, and the symptoms those of a profound cerebral neurasthenia, with gastrosuccorrhea. The autopsy showed dilation of the stomach and chronic catarrhal gastritis. The microscope revealed a frayed-out mucous membrane; beneath this, a cellular tissue quite rich in cells, with tubules containing more or less well-preserved epithelium, the tubules not reaching more than about half-way into the depth of the mucous membrane; below this there was more cellular tissue, showing no longitudinal sections of tubules, but tubules in nest-like aggregates, containing well-preserved and proliferated epithelium. As to the nature of these newly formed cells, whether they were chief cells or parietal cells, the authors do not decide, although they seem to be inclined to look upon them as being the latter. They believe that the lesion found by them explains the intermittent gastrosuccorrhea. They assume that through the influence of the nervous system there occurred an increased functional activity of the epithelium, which in turn led to a hyperplasia of those cells particularly which secrete hydrochloric acid. The gastric juice, which was thus secreted in excess, was poured out over the stomach, causing an irritation which eventually led to interstitial inflammation.

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[1] Soc. med. Chir. di Modena, April 19, 1899; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[2] Compt. rend. de la Soc. de Biol., April 6, 1900.

[3] Centralbl. f. allg. Path. u. pathol. Anat., Feb. 15, 1900.

[4] Virchow's Archiv, Bd. CLVII, Heft 1, p. 1.

**False Diverticula of the Bowel.**—Graser[1] attributes the false diverticula of the sigmoid flexure to venous stasis. These diverticula may lead to complications, in that fecal stones remaining in them may produce atrophy of the mucous membrane, and even perforation into the fatty tissue; and he believes that certain cases of chronic mesenteric peritonitis of the sigmoid flexure are to be attributed to such diverticula.

**Hemorrhagic Infarction of the Small Intestine.**—Le Wald[2] reports a case of hemorrhagic infarction of the small intestine and mesentery, due to thrombosis of the superior mesenteric artery. Perforation of the intestine occurred, with general peritonitis. There was extensive atheroma and calcification of the artery and dilation of the heart. The kidneys were much enlarged and there was an infarct in one of them. The infarcted and gangrenous portion of the intestine was 5 inches long.

**The Pathology of Appendicitis.**—A. O. J. Kelly[3] discusses the pathology of appendicitis based upon the systematic examination of 577 appendices removed at operation and upon considerable necropsy experience. The following divisions of the subject are considered seriatim: (1) The lesions of the appendix; (2) the peritonitis and its consequences; (3) the bacteriology; and (4) the pathogenesis. In discussing the lesions of the appendix the following classification is adopted: (I) Acute appendicitis—(1) catarrhal, (*a*) simple, (*b*) purulent, (*c*) hemorrhagic; (2) interstitial; (3) ulcerative, (*a*) nonperforative, (*b*) perforative; (4) gangrenous. (II) Chronic appendicitis—(1) catarrhal; (2) interstitial; (3) obliterating. The macroscopic and microscopic lesions of each of the varieties of appendicitis, as well as those of tuberculosis and actinomycosis of the appendix, are described in detail. In discussing appendicular peritonitis and its consequences the pathogenesis of the condition is discussed, and the following classification adopted: (I) Acute appendicular peritonitis—(1) circumscribed serous, serofibrinous, and fibrinous peritonitis; (2) circumscribed purulent peritonitis or periappendicular abscess; (3) diffuse or generalized peritonitis. (II) Chronic appendicular peritonitis. The pathologic anatomy of each is described as well as the varying association of the different forms of peritonitis with various forms of appendicitis. Of the diffuse peritonitis, the following are described: The progressive fibropurulent peritonitis of Mikulicz; the diffuse purulent or suppurative peritonitis; putrid peritonitis; hemorrhagic peritonitis; and septic or toxic peritonitis. In discussing the bacteriology of appendicitis it is stated that of the 286 cases examined bacteriologically, Bacterium coli commune was found alone in 72.65% of the acute cases and in 89.873% of the chronic cases; and that it was found either alone or in combination with other bacteria in 91.4% of the acute cases and 96.2% of the chronic cases. Predominating importance in the production of appendicitis is accorded Bacterium coli commune, but the significance of other bacteria, such as the staphylococci, the streptococci, Bacillus pyocyaneus, etc., is not underestimated. In discussing the pathogenesis of appendicitis (see also the "Philadelphia Medical Journal," vol. IV, 1899, pp. 928, 983, 1032) the following causes predisposing to inflammation of the organ are discussed: (1) The shape of the meso-appendix; (2) the excessive length as compared with the caliber of the lumen; (3) Gerlach's valve; (4) the histologic structure of the organ; (5) the blood supply; (6) the nerve supply; and (7) the evidences of involution of the organ. The significance of traumas, foreign bodies, and appendicial calculi is also considered in detail. The most important factor in the pathogenesis of appendicitis is defective drainage, which leads to increase in the virulence of the contained bacteria; the occurrence of this is facilitated by various anatomic and physiologic peculiarities of the appendix, as well as by the consequence of previous disease.

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[1] Langenbeck's Archiv, No. 59, 638, 1899; Centralbl. f. allg. Path. u. pathol. Anat., Aug. 24, 1900.

[2] N. Y. Path. Soc., Nov. 8, 1899; Med. Rec., Dec. 3, 1899.

[3] A Treatise on Appendicitis, by John B. Deaver, p. 41, 2d ed.; P. Blakiston's Son and Co., 1900.

**Tumors of the Vermiform Appendix.**—A. O. J. Kelly,[1] in a microscopic examination of 706 appendices removed by operation, detected 3 instances of adenocarcinoma and one of endothelioma. In 3 of the cases the growth was primary in the appendix, and probably also in the fourth instance. The tumors were of microscopic size and were not detected by examination by the unaided eye. The youth of two of the patients, who were aged 24 and 19 respectively, is also of interest.

Ssobolew,[2] reporting a most unusual case of **endothelioma of the gastro-intestinal tract,** states his belief in the necessity of designating a class of tumors as endotheliomas because of the following: (1) The endothelium, to a certain degree capable of secretion, is closely allied physiologically to epithelium; (2) endothelial cells resemble epithelial cells; they possess but slight disposition to the formation of intracellular substance; (3) in various disease processes they give rise to very peculiar products, as, for instance, in endotheliomas of the meninges; and (4) the histogenesis of endothelium has not yet been explained. The case reported is that of a woman aged 28 years, who, after an illness lasting about a year, died, the clinical diagnosis being carcinoma of the stomach with adhesions to the right kidney and the ascending colon. At the necropsy there were found throughout the entire gastro-intestinal tract numerous nodules the size of peas and smaller. On microscopic examination these were recognized as endotheliomatous, and the portions of the tract unaltered to the unaided eye were found to be also the seat of endotheliomatous new formation. The endotheliomatous origin of the tumor was evident from the arrangement of the cells into bands coursing in the direction of the surrounding fibers, the absence of proper stroma, the transition forms from normal endothelium to tumor-cells, the resemblance of the latter to epithelial cells, the intimate relationship—at times by means of prolongations—with the fibers and cells of the surrounding connective tissue, and the fact that the tumor-cells bore no relationship to proliferated glandular epithelium. The peculiarities of the tumor consisted in the multiplicity of the new formations, the relations of the cells to the stroma,—especially the formation of intercellular substance from the latter,—and the presence of intracellular vacuoles attributed to fat and possibly glycogen.

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[1] Proc. Path. Soc. of Phila., Mar. 19., 1900; A Treatise on Appendicitis, by John B. Deaver, 2d ed., p. 95, P. Blakiston's Son and Co.; Univ. Med. Mag., May, 1900.

[2] Virchow's Archiv, vol. CLXI, p. 56, 1900.

**Anemic Infarction of the Liver.**—Owing to the abundant supply of blood from several sources, the liver is seldom the seat of infarction. At one time, indeed, it was believed that the process did not occur in the organ at all. Lazarus-Barlow [1] describes an interesting case of such infarction. The patient had been crushed between the buffers of two railway cars. There was a large rupture in the right lobe of the liver, and near the middle of the rupture there was a pale-yellow mass of tissue, ovoid superficially and wedge-shaped on section. It was 2 inches in length, 1½ inches in greatest width, and 1 inch in depth. Hepatic infarcts rarely depend upon embolism of the hepatic artery, and even in thrombosis of the veins in the intestinal area infarction is not common. In a large proportion, however, the portal vein or its branches are involved. Chiari in 17 cases found 15 dependent upon embolism and 2 upon thrombosis of large branches of the portal vein; in a case of Osler's there was a thrombus in the portal vein. But thrombosis of large portal branches is met without coexisting hepatic infarct. Chiari suggests that the effective adjuvant to portal obstruction in the causation of hepatic infarct is a very low blood pressure, and this view is shared by Lazarus-Barlow. Regarding the relative frequency of hemorrhagic and anemic infarction, the former seems to be the more common. Including the case observed by Barlow, there are now 30 instances of infarction of the liver on record. It is probable that a careful research for hepatic infarcts might show them to be more frequent than has hitherto been believed.

**The Intracellular Radicles of the Biliary System and Icteric Necrosis of the Liver Cells.**—Fütterer [2] was enabled to study the radicles of the biliary system, and icteric necrosis of the liver-cells in a liver the seat of carcinoma. The carcinoma had implicated and induced closure of the hepatic duct, so that in consequence there occurred complete stasis of the bile and natural injection of the biliary radicles. His conclusions are as follows: (1) The radicles of the biliary system are situated in the liver-cells as intraprotoplasmic channels which surround the nucleus especially, and form a protoplasmic network; (2) there does not appear to be an intranuclear channel-system communicating with the intraprotoplasmic system; (3) the intraprotoplasmic channels are in direct communication with the biliary capillaries; (4) under normal conditions the intraprotoplasmic channels are not visible, and when under pathologic conditions (bile stasis) they become visible, this ensues at the expense of the substance and life of the liver-cells; (5) while the cell protoplasm, under these conditions, rapidly degenerates, the nucleus remains intact for an extremely long time; (6) the bile is secreted within the liver-cells in the form of minute droplets in the intraprotoplasmic biliary network and collects first in the neighborhood of the nucleus.

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[1] Brit. Med. Jour., Nov. 11, 1899.

[2] Virchow's Archiv, vol. CLX, p. 394.

**Liver Lymphomas in Infectious Diseases.**—Marcuse[1] details the results of an investigation of 114 cases of various infectious diseases with respect to the occurrence of lymphomas of the liver. By lymphoma is understood, according to Virchow, whose definition is accepted, a collection of closely compressed, uninuclear, round cells (lymph-cells) contained in larger or smaller foci in a fine connective-tissue reticulum. Such lymphomas of the liver are frequent in diphtheria and scarlet fever, and in typhoid fever also. They are less common in measles, pneumonia, and other infectious diseases. The normal interstitial connective tissue of the liver, as a matter of fact, contains more round cells in childhood than in adult life. But the occurrence of typical and characteristic lymphomatous proliferations in the liver is dependent upon the diseased process and not upon the age of the individual. The lymphomas are demonstrable a few days after the commencement of the disease; they persist a few days after the disease, and disappear gradually without necrobiotic or necrotic processes. As they disappear there seems to occur a proliferation of the preformed connective tissue. In none of the cases the subject of examination did it seem that the lymphomas occurred only at a certain stage of the disease. Nor in any of the cases was there demonstrable a connection between the occurrence of the lymphomas and the development of certain anatomic findings, such as swelling of the intestinal follicles, hemorrhagic nephritis, hemorrhages into the mucous membranes, etc., although one of these—the alterations of the intestine—in the cases investigated was very common. The largest lymphomas measured 300 $\mu$ in diameter; they were situated especially at the points of division of the biliary ducts or the portal veins, which the lymphomatous proliferations surrounded as a hollow cylinder. In many places the lymphomatous tissue replaced all interstitial liver tissue, so that fine fibrous tissue was found only at the unaffected external layers of the capsule of Glisson and about the large branches of vessels. The walls of the arteries, as also those of the central veins, remained free from round-cell infiltration. Small lymphomatous foci situated in the center of the acini were in relation with the portal tissue. Necrosis of the liver was not to be found within the real lymphomas. The lymphomas have nothing whatever to do with the circumscribed foci of necrosis to be observed not only in typhoid fever, but also in scarlet fever and diphtheria.

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[1] Virchow's Archiv, vol. CLX, p. 186, 1900.

**Cirrhosis and Multiple Adenoma Formation of the Liver.**—Schmieden,[1] after a critical discussion of the previous literature on cirrhosis and adenoma formation in the liver, reports in elaborate detail a personal observation. The liver was slightly enlarged, its surface granular, its consistency increased; on section it was granular, and presented a number of prominent yellowish-white areas, and a large amount of connective tissue pervading the entire organ. Microscopically the cirrhosis was found to be of the annular variety; the connective tissue was poor in nuclei. Weigert's stain revealed the presence of a large number of elastic fibers. The liver acini were markedly atrophic and degenerated and reduced to at least one-fourth of their normal size and number. There was, however, distinct evidence of hypertrophy and proliferation of the liver-cells, with resultant giant cells (some containing as many as 20 nuclei), and regular and irregular karyomitotic figures. In intimate association with these areas of hypertrophic liver-cells were the yellowish-white nodules, evident upon macroscopic examination. These revealed transition from the large liver-cells, some with many nuclei, to columns of adenomatous formation. The commencement of the tumor-formation varied in different sections, but it was not confined to any one particular portion of the liver acini. The cells revealed many mitotic figures, and as the nodules increased in size they caused pressure necrosis of the adjoining liver-cells. It is believed that the changes observed constitute a series of related processes; that the oldest and primary alteration was the cirrhosis of the liver; in consequence of this, and to replace the destroyed liver-cells, a vicarious hypertrophy of remaining liver-cells resulted, and from this the adenoma formation, and ultimately a malignant metamorphosis. In this way an autochthonous primary carcinoma of the liver due indirectly to the cirrhosis was brought about.

**Primary Carcinoma of the Ductus Choledochus.**—Brenner[2] reports two cases of carcinoma of the ductus choledochus, both of which had given metastasis to the liver and lymphatic glands. Contrasting these cases with those found in the literature, he finds that there is no seat of predilection for the growth. In 8 cases the tumor was seated at or near the papilla; in 2 cases at the junction of the hepatic and cystic ducts; in five other cases and in the two reported it was situated between this juncture and the exit of the duct into the intestine. In general the tumors are characterized by their small size; they are generally described as being the size of a cherry, hazelnut, or walnut. One of the two reported by Brenner was the size of a hen's egg, and this is probably as large as any described. In form the carcinoma may occur either as a flat infiltration or as a more compact papillomatous growth (such as Brenner's first case). In general the infiltrating form does not reach a greater size than does the tuberous, but it may, as in the second of Brenner's specimens. The point of departure of the growths is the epithelium of the glands of the duct. In most cases, however, there is marked implication of the connective tissue, so that the neoplasms have been described as scirrhous. In all the reported cases there was closure of the ductus choledochus, stasis of the bile, and dilation of the biliary passages. As a consequence of the congestion of the bile, in most cases continuing for months, changes described as biliary cirrhosis ensue in the liver. This may reach a high grade and result in the production of enlargement of the spleen and ascites. If the growth be situated near Vater's papilla, it may cause obstruction of the flow of the pancreatic juice and dilation of the pancreatic duct, as was observed in one of Brenner's cases. Usually carcinoma of the ductus choledochus does not give rise to metastasis; in 10 of the reported cases there were no secondary formations. The clinical diagnosis of the condition is impossible, but the disease may be suspected if the symptoms can be referred to closure of the ductus choledochus, and the patient be elderly and cachectic. With these there may be associated symptoms on the part of the biliary system, such as biliary colic, fever, vomiting, and diarrhea, and other indications of gastro-intestinal catarrh. At times a tumor may be palpated. Exploratory operation may facilitate the diagnosis. As regards the etiology of the tumor formation, the relationship of cholelithiasis, which has been observed in some cases, is considered; but in view of the absence of gallstones in most of the cases it does not possess much significance.

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[1] Virchow's Archiv, vol. CLIX, p. 290, 1900.

[2] Virchow's Archiv, vol. CLVIII, p. 253, 1899.

**Pancreatitis.**—Flexner [1] injected into the pancreatic duct of dogs weak acids, weak alkalies, formalin, and cultures of the diphtheria bacillus and of Bacillus pyocyaneus, and obtained by all these methods a pancreatitis—most successfully, however, by the injections of hydrochloric acid. The forms of pancreatitis were hemorrhagic, necrotizing, and suppurative. Sugar was found in the urine of animals which succumbed to the acute lesions. Fat necrosis also occurred. An examination of the latter lesions demonstrated the presence of the fat-splitting ferment, thus disposing of the assertion of Hlava that the fat necrosis is not due to the pancreatic ferment. Histologically the pancreatic lesions were in every way comparable to those of man. While the conditions of the various experiments were such that they would hardly find their duplication in men, it is, nevertheless, reasonable to infer that human pancreatitis is of bacterial origin. It is not probable that enough gastric juice could escape into the pancreas through its duct to set up an inflammation. The fat necrosis is unquestionably due to the action of escaped pancreatic ferment.

**A Macrochemic and Microchemic Reaction for Fat Necrosis.**—Benda [2] found accidentally that preparations from a case of fat necrosis, having been hardened in 10% formalin solution and placed for 24 hours in Weigert's neuroglia stain, presented an appearance as though covered with verdigris. Section of the preparation revealed the same appearance throughout. Subsequent investigations disclosed that a like result was achieved with the use of the copper acetate solution alone (2.5 % aqueous solution). The greenish-blue color was sharply circumscribed to the necrotic foci, and permitted the detection (in the pancreas) of foci invisible to the unaided eye. Microscopically normal fat-cells presented no trace of blue color. In the necrotic foci the stratified masses composed of calcium salts of the fatty acids were stained a pale greenish-blue ; the most intense greenish-blue color, however, was presented by the acicular fatty acid crystals. The color persisted in sections preserved 4 weeks. Benda attributes this reaction—the occurrence of a greenish-blue color in formalin preparations—to the combination of fatty acids, especially oleic acid, with copper oxid, and believes the test a delicate macrochemic and microchemic reaction for fat necrosis. It permits of the recognition of the earliest stages of the necrosis, which he believes are independent of inflammatory processes.

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[1] Proc. Path. Soc. of Phila., Dec. 1, 1899.

[2] Virchow's Archiv, vol. CLXI, p. 194.

## THE RESPIRATORY TRACT.

**Bacteriology of Ozena.**—A series of investigations leads De Simoni [1] to the following conclusions : (1) Pathogenic bacteria, such as are occasionally found in the normal nasal mucus, are constantly found in ozena. These bacteria are the diplococcus of Fraenkel and the pyogenic staphylococci and streptococci. (2) The nonpathogenic bacteria constantly found in ozena, and occasionally in the normal mucus, are encapsulated and diphtheria-like bacilli. (3) The pathogenic bacteria which are constantly found in ozena have no etiologic significance. As thus the infectious nature of ozena is excluded, it must be assumed that the cause of the condition is a primary anatomic lesion of the nasal mucosa which permits the multiplication of pathogenic and nonpathogenic bacteria which contribute to damage the function of the epithelial cells.

The same author [2] has studied the so-called mucosus bacillus of ozena, and comes to the conclusion that the different varieties described may be divided into 3 groups, the type-form of which is the pneumobacillus of Friedländer, which is a habitual denizen of the nasal and pharyngeal mucosa. Physical as well as biochemical influences can transform one variety into another.

Fernand Perez [3] thus describes the principal characteristics of the Coccobacillus fœtidus ozenæ : It is immobile, does not stain by Gram's method, does not liquefy gelatin, does not cause fermentation in solutions of lactose, and never coagulates milk. It produces indol, ferments urea, and is pathogenic for guinea-pigs, mice, pigeons, and rabbits. Nearly all its cultures give off a characteristic fetid odor. The infection that it produces in the rabbit may be acute, subacute, or chronic. In all forms of infection the organism, which is not very virulent, produces at the beginning a very intense nasal discharge, which is sometimes hemorrhagic; indicating an elective action upon the nasal mucous membrane. At autopsy animals that have died after intravenous injection of the organism do not present the generalized lesions of hemorrhagic septicemia, and the principal, almost the sole, lesion is a hyperemia, often hemorrhages, of the pituitary mucous membrane, which is covered by a thick mucus in which the inoculated organism may be found. In the chronic forms of the infection the animals present the characteristic atrophy of ozena. The author concludes that Coccobacillus fœtidus ozenæ is the specific micro-organism of atrophic fetid rhinitis.

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[1] Il Policlinico, Aug. 15, 1899 ; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[2] Centralbl. f. Bakt., Parasit. u. Infektionskrankh., April 20, 1900.

[3] Ann. de l'Inst. Pasteur, Dec., 1899.

**Emphysema of the Lung.**—There are two principal theories to explain emphysema of the lung: the one, the mechanical theory; and the other, the theory that the disease is due to nutritive disturbances in the lung tissue. Eppinger, an adherent of the mechanical theory, attributes great importance to the elastic tissue, which he believes becomes diminished in emphysema. Sudsuki[1] has undertaken a study of the elastic tissue in emphysema, using Weigert's method of staining. A total of 45 cases was examined, of which 30 were substantial, 13 vicarious, and 2 acute emphysema. The observations were compared with those on the elastic tissue in normal lungs. The results showed, in the first place, that there was an individual difference in the amount of elastic tissue in the alveolar walls, but that neither sex nor age produced any variation. In the emphysematous lungs there was no actual diminution of the elastic tissue. Where the lung tissue was destroyed by the emphysema, the elastic tissue had also suffered; but in the preserved portion of the lung the elastic tissue was undiminished. The apparent diminution of the coarser and medium elastic fibers in emphysematous air vesicles was relative, and depended upon the fact that the elastic tissue is distributed over a greater area than normal. Through ligation of the trachea in animals the author produced an emphysema which in every way corresponded to that of the human subject. There was no actual diminution in the elastic tissue here, but only a relative thinning from overstretching. The author, very naturally, having been a pupil of Hansemann, is a believer in the existence of pore canals in the lung. These pore canals are greatly dilated in emphysema. His final conclusion regarding emphysema is that it is due to mechanical causes, and that imperfect development or diminution in the elastic tissue plays no etiologic rôle. The possibility of a functional weakness of the elastic tissue can not be determined by the pathologist. On account of the repeated mechanical dilation, the alveolar walls lose their elasticity, like an oft-stretched rubber band. It is possible that certain inflammatory processes or some congenital conditions cause an impairment in the elasticity of the lung, and that this favors emphysema. The dilation of the stomas causes changes in their edges, and eventually a confluence of contiguous stomas, producing large defects in the partition walls of the alveoli.

**Clinical Application of the Histologic Study of Serofibrinous**

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[1] Virchow's Archiv, Bd. CLVII, p. 438.

**Pleural Effusions.**—Widal and Ravaut[1] have studied 56 cases of serofibrinous effusion in the pleura. As a result of their observations they are able to say that the study of the cells found in these effusions gives valuable clinical data. By an exploratory puncture with a sterile syringe, several cubic centimeters of the effusion are withdrawn. The fluid is then to be defibrinated by shaking it at once with glass beads. Then the fluid is to be decanted and centrifuged. The cellular elements will then be found in the bottom of the centrifuge tube and can be taken out and placed on a slide. The sediment may be stained with thionin, hematoxylin and eosin, and the triacid mixture of Ehrlich. Idiopathic pleurisy, which is nearly always tuberculous, is characterized by the presence almost exclusively of lymphocytes, very confluent and frequently mixed with a relatively considerable number of red cells. There are also a few elements that may be large mononuclear elements or endothelial cells, and amorphous masses without a nucleus. In only 2 cases were micro-organisms found. The lymphocytosis is the characteristic feature of these effusions. The fluid from cases of tuberculous hydropneumothorax presents old, polymorphonuclear leukocytes that are deformed and contain altered neutrophilic granulations. The mechanical pleurisies developing in the course of heart disease, Bright's disease, or carcinoma, or by compression or irritation, are characterized by the presence of endothelial cells, which may be recognized by staining with hematoxylin and eosin. In the fluid from a case of streptococcic pleurisy polymorphonuclear neutrophiles with deformed nuclei were found, together with the streptococci. In pneumococcic pleurisy the elements found were red cells, some lymphocytes, abundant polymorphonuclear leukocytes, and macrophages containing the polynuclear leukocytes in their protoplasm. Typhoid pleurisies are often hemorrhagic, and the presence of blood obscures the histologic characteristics of the effusion, which is composed of large mononuclear leukocytes, with the occasional presence of eosinophiles. Three cases of pleural effusion in which there were numerous eosinophile cells are recorded. One of these was in a case of typhoid pleurisy, another was in a case accompanying a probable parasitic affection of the liver, and the third was in a tuberculous patient.

**The Histology of Acute Lobar Pneumonia.**—Pratt[2] summarizes his observations as follows: Early in the disease the alveoli contain many cells almost identical in appearance with the transitional cell of the blood. Their origin is uncertain, but it is probable that they arise from proliferation of the cells lining the alveoli. They may be transitional leukocytes that have migrated from the blood-vessels. Similar cells are found in the lymphatics, blood-vessels, and interstitial tissue of the lung, in the pleural exudate, and in the bronchial lymph-nodes. Large phagocytic cells are found in all stages of the disease; in greatest number in gray hepatization; they probably arise from the alveolar epithelium, and resemble the phagocytic endothelial cells described by Mallory in typhoid fever. It is very likely that they play an important part in resolution. Similar phagocytic cells occur in the lymphatics, the pleural exudate, and the bronchial lymph-nodes. The fibrin comes exclusively from the blood. The lymphatics are involved late in the disease. There is proliferation of their endothelium, and they become distended with cells, serum, and fibrin. Early in the disease there is no infiltration of the interstitial tissue, but in cases dying during the second week there is often a great infiltration with lymphoid and plasma cells. As a rule, the longer the duration of the disease, the greater the number of plasma cells.

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[1] Compt. rend. de la Soc. de Biol., July 6, 1900.

[2] Contributions to the Sci. of Med., W. H. Welch's Festschrift, p. 265.

**Variations of the Iodin in the Thyroid Body under Pathologic Influences.**—Charrin and Bourcet [1] have studied the thyroid bodies of infants from 1 day to 3 months old. The absence of iodin from the thyroid was observed in those cases in which the mothers and the babies were either ill or had suffered from previous disease. When the mother was alcoholic, or had typhoid fever, malaria, pneumonia, or tuberculosis toward the end of gestation, and the child presented gastro-enteritis or bronchopneumonia, it was not rare to find iodin diminished in quantity or absent.

## THE NERVOUS SYSTEM.

**Acute Spinal Perimeningitis.**—Acute spinal perimeningitis is an inflammation of the cellulo-adipose tissue surrounding the spinal dura mater and separating it from the vertebræ. Delearde,[2] in connection with a case observed by himself, has made a study of the condition. The term should be limited to primary inflammation of the cellulo-adipose tissue, and should not be extended to include secondary inflammation induced by invasion from the vertebræ, muscles, or other adjacent parts. There are 16 cases recorded in the literature of the subject, most in men, exposure and hard labor playing an important etiologic rôle. Since the dura mater is adherent at its anterior surface to the posterior surface of the vertebræ, through the intermediation of the posterior vertebral ligament, and is separated from the other portions of the vertebræ by a cellular tissue rich in fat, the purulent collections of perimeningitis localize themselves on the lateral and posterior aspects of the membrane, between it and the lamellæ of the vertebræ. In nearly all the cases the dura in contact with the pus is somewhat injected, at times covered with false membranes. The spinal cord was softened at the level of the inflammation. [Symptomatology and diagnosis do not come within the scope of this abstract.]

**Acute Streptococcal Meningitis Supervening in the Course of Chronic Parenchymatous Nephritis.**—Rolleston [3] reports a case of extensive purulent meningitis due to streptococcal infection, associated with an acute nephritis that had been superadded to a chronic parenchymatous Bright's disease. The case was one of terminal infection of the meninges, and is interesting, since such infections in renal disease are more common in the lungs, pleura, and pericardium. The tonsils (there had been a tonsillitis) may have been the source of infection.

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[1] Compt. rend. de la Soc. de Biol., April 6, 1900.

[2] Gaz. hebdom. de méd. et de chir., May 27, 1900.

[3] Brit. Med. Jour., Oct. 21, 1899.

**The Ependyma of the Cerebral Ventricles in Tuberculous Meningitis.**—Walbaum,[1] as a result of the investigation of 28 cases, has been able to confirm the previous observations of Ophüls,[2] that in almost all cases of tuberculous disease of the meninges the ependyma is involved, and that the lesions here are also tuberculous. In some of the cases the granulations were not discernible macroscopically, and at times careful and patient investigation of the entire lining and all the recesses of the ventricles was necessary to discover the lesions. In the majority of cases, however, they were immediately patent, and manifested no special predilection for any particular locality. In cases in which they were rather sparse they involved chiefly the dependent portions and the folds, just as is the case in granular ependymitis of nontuberculous nature. In tuberculous meningitis 4 varieties of granulations may be found in the ependyma: (1) Nodules indicative of a concomitant granular ependymitis; (2) nodules similar to the preceding, but into which tubercle bacilli have wandered secondarily, and in which sometimes slight increase of the round cells may be detected; (3) superficial collections of cells produced by the invasion of tubercle bacilli from the ventricular cavity; and (4) formations situated deep in the ependyma and recognizable as tubercles. The last two only are characteristic of tuberculous ependymitis. Tubercle bacilli are usually detected with ease. The glia cells are only passively involved, and in consequence of the progressively enlarging collection of cells they gradually degenerate. Proliferation of the lymph-vessel endothelium is common.

**Giant Growth and Tumor of the Pineal Body.**—Oestreich and Slawyk[3] report the case of a male child, aged 4, who, previously healthy and lively, at the age of 3 became quiet, shy, and retiring, and cried considerably. At this time his body commenced to grow rapidly, especially so the penis. During the 4 weeks prior to first observation his gait became poor, and he was subject to attacks of psychic depression and abstraction, during which he could not be made attentive and refused to answer questions. Examination revealed hypertrophy of the breasts, each of which was 2 cm. high; enlargement of the penis, which measured 9 cm. in length in the relaxed state; his testicles the size of hen's eggs; a considerable growth of hair about the pubes; unsteady and somewhat spastic gait; exaggerated superficial and deep reflexes; choked discs; and slow, irregular pulse. In the absence of evidences of hydrocephalus the diagnosis of brain tumor was made, and as there were no focal symptoms, the growth was thought to be situated in the median line or in the white matter. Although typical signs of acromegaly (enlargement of the extremities and of the face) were absent, it was thought that the giant growth and the brain manifestations were probably related to each other, and a provisional diagnosis of tumor of the hypophysis was made. At the end of five weeks the patient died with convulsions and in coma. The necropsy revealed a cystic psammosarcoma of the pineal body, with internal hydrocephalus, the hypophysis being normal. Although no analogous case could be found in the literature,—all the tumors of the pineal body giving rise to general brain symptoms and manifestations on the part of the eyes,—it is believed that diseases (tumors) of the pineal body may give rise to anomalies of growth, similarly as do disorders of the hypophysis result in the production of acromegaly. And the analogy is further borne out by the fact that as only certain diseases of the hypophysis are associated with acromegaly, so also may only certain diseased conditions of the pineal body result in the production of giant growth.

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[1] Virchow's Archiv, vol. CLX, p. 85, 1900.

[2] Virchow's Archiv, vol. CL.

[3] Virchow's Archiv, vol. CLVII, p. 475, 1899.

**The Pathology of Tabes Dorsalis in Relation to General Paralysis of the Insane.**—An interesting discussion, participated in by Mott, Bruce, Buzzard, Gowers, Savage, Ferrier, Payne, Hale White, and Batten, took place before the London Pathological Society on November 21 and December 5, 1899.[1] Mott stated (1) that it was well known that a certain number of cases of tabes might begin with mental symptoms, or that mental symptoms in the form of crises might occur in the course of tabes; (2) that a number of cases of general paralysis present tabetic symptoms during life, and postmortem show atrophy and sclerosis of the posterior columns of the cord and the posterior roots; (3) that a few cases had been met which later developed well-marked symptoms of general paralysis. Syphilis was the most important factor in the production of both diseases, and in 22 cases of juvenile paralysis—one of which was of the tabetic type—that he had seen, a definite history of syphilis was obtained in 60%, and could not be excluded in the remainder. A number of authorities have examined the spinal cord in the tabetic form of general paralysis, some claiming to find lesions of the same nature as tabes, while others held that they were different. In the cases examined by Mott the lesions were typical of tabes and the brain changes were those of general paralysis. Mott had examined the spinal ganglia in 6 cases, and in only 2 were there definite chromolytic changes. In both of these the peripheral nerves were affected. In all four cases, however, the cells showed excessive pigmentation and some shrinking and irregularity.

Alexander Bruce discussed the pathology of tabes, and rejected the hypothesis of Marie that the degeneration is due to nutritive changes in the cells of the posterior root ganglia, and adopted that of Obersteiner: namely, that the action of the nutritive center in the ganglion of the posterior root was cut off by pressure upon the fibers at the site of entrance into the spinal cord. The normal constriction occurring at this point might be increased by extrinsic causes, such as meningeal thickening, the result being that the posterior root between the ganglion and the cord remained intact, while the degeneration involved the strictly intraspinal fibers. Such meningeal thickening might be syphilitic or due to other causes.

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[1] Brit. Med. Jour., Nov. 25 and Dec. 9, 1899.

**The Pathology of Sleeping Sickness.**—The cause of the African sleeping sickness is not known. It is always fatal, and in 2 cases reported by Manson and Mott[1] death was due in the one to hyperpyrexia, and in the other to a series of epileptiform seizures. The blood in this disease always contains Filaria perstans, but as this is a common parasite in the blood of the natives, it could not be considered the cause of the disease. Microscopic examination was made by Mott in Manson's two cases—the brain, the spinal cord, the pituitary body, and the spinal ganglia being examined. There was found a leptomeningitis and an encephalomyelitis. The perivascular spaces were distended with mononuclear leukocytes. Bacteria were not found on staining. The ganglion cells themselves throughout the whole nervous system showed a uniformly dull staining reaction, and in none of the cells were the Nissl granules evident. This case was undoubtedly due to the hyperpyrexia during the last hours of life. In the second case the cells for the most part presented a normal outline and exhibited Nissl granules. In the medulla, however, a considerable number of cells showed chromolytic changes, and similar changes of a less degree were found in the motor cells of the anterior cornea. Regarding the nerve-fibers, there was some wasting of the tangential fibers of the cortex in one case and a decided wasting in the other. There was no endarteritis. The central canal of the spinal cord was filled with proliferated pia tissue. The posterior spinal ganglia showed the vessel changes described, but the ganglion cells in the first case showed only the diffuse staining of hyperpyrexia, and in the second exhibited a fairly normal appearance. The symptoms of the disease—namely, progressive drowsiness and lethargy and progressive weakness in body and mind, without any distinct paralysis or mental disability—can best be accounted for by supposing that the metabolism or functional activity of the neurons, as a whole, was affected injuriously by some toxic product circulating in the blood or existing in the cerebrospinal fluid; that this toxic agent occasioned great proliferation of the mononuclear leukocytes of the brain and pia lymphoid, and in the perivascular lymphatics. The liver, kidneys, lungs, pituitary body, spleen, lymph-glands, and duodenum were also examined; the results obtained were, for the most part, with the exception of the duodenum and lymphatic glands, negative. The lymph-glands were much enlarged, owing to a great increase of lymphocytes; sections of the duodenum showed a large number of lymphocytes and a proliferation of the same in the lymphoid nodules.

**Histologic Changes in the Nervous System in Uremia.**—Ciauri[2] found the most marked changes in the cerebrum, the changes varying all the way from a simple chromatolysis to the formation of vacuoles. Golgi's method revealed no changes.

**Changes in the Central Nervous System in Experimental Cholemia.**—Malfi and Antinori[3] produced cholemia by ligation of the common bile-duct in rabbits and dogs. Golgi's method showed no changes in the nervous system. By Nissl's stain they found chromatolysis and changes in the nucleus in the large cells of the motor area.

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[1] Path. Soc. of London, meeting of Oct. 2, 1899; Brit. Med. Jour., Oct. 21, 1899.

[2] Tenth Ital. Cong. for Intern. Med.; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[3] Riforma med., 1899, I, 33; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

**The Influence of Fatigue on the Nerve-cells of the Cortex.**—Guerrini[1] caused dogs to walk many kilometers by means of a special apparatus, and studied the nerve changes in the cerebrum and cerebellum. They consisted in enlargement of the pericellular space and apparently of leukocytosis in that space; alterations in the chromatic substance, with disintegration of the Nissl bodies, and the appearance of vacuoles; irregularity and vacuolization of the nucleus.

**The Staphylococcus and Rheumatic Chorea.**—According to Mircoli,[2] rheumatic chorea is an infectious disease due to various bacteria, the most important being the staphylococcus.

**The Presence of Staphylococci in Sydenham's Chorea.**—Although Guidorossi and Guizzetta[3] found the staphylococcus in the blood and in the organs in a case of chorea, they conclude that it probably bears no relation to the disease, and represents only a secondary infection.

**The Pathology of Sydenham's Chorea.**—In a case of chorea Daddi and Silvestrini[4] found alterations in the cells in the cortex, consisting in a chromatolysis and a peripheral dislocation of the nucleus. In the cerebellum the lesions were mild. In the medulla chromatolysis was of the highest degree. In the spinal cord the ganglion cells showed degenerative changes in Nissl's bodies. Similar changes were found in the spinal ganglia.

**The Pathologic Anatomy of Idiocy.**—Schutte[5] publishes a magnificent digest of the most important publications on this subject, in which he deals with 339 separate articles.

**The Changes in the Spinal Cord, Spinal Roots, and Peripheral Nerves in the Status Epilepticus.**—Cristiani[6] found in the spinal cord changes in the cells, and in the spinal roots and peripheral nerves the features of a parenchymatous neuritis.

**Guinea-pig Epilepsy and Its Hereditary Transmission.**—Brown-Séquard found that epileptic convulsions occurred in guinea-pigs after any one of the following injuries: (1) Complete or almost complete unilateral section of the spinal cord; (2) simultaneous section of the posterior columns, the posterior horns, and a portion of the lateral columns; (3) division of both posterior columns, of the lateral columns, or of the anterior columns; (4) complete division of the cord; (5) simple puncture; and (6) section of the sciatic nerve. In guinea-pigs treated in any one of these various ways irritation of certain epileptogenic zones produces convulsions. Similar experiments were made by Westphal and Obersteiner, who were able to confirm Brown-Séquard's observations. The latter also maintained that the offspring of epileptic animals were epileptic and had certain defects of the toes corresponding to those produced in the parents by division of the sciatic nerve. Sommer [1] has repeated Brown-Séquard's experiments in 40 guinea-pigs, and found that epileptic convulsions occurred in all the animals; but he looks upon them, not as genuine epilepsy, but as reflex epilepsy, and does not believe that they can be compared with idiopathic epilepsy in man. These 40 animals gave birth to 23 young, in none of which epilepsy developed, nor were the defects of the toes of the parents produced by division of the sciatic nerve transmitted to the offspring. [Although the question of the hereditary transmission of acquired characters has been for years one of great interest, it still remains unsettled. Among pathologists, Ziegler is one of the strongest adherents of Weissmann's view that pathologic characters are not transmitted. The burden of proof rests upon those who claim that they are transmitted; the negative experiments of Sommer are of value, in that they disprove the validity of one of the famous objections to the Weissmann theory.]

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[1] Riforma med., 1899, II, 26; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[2] La clin. med. Ital., No. 4, 1899; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[3] Riforma med., 1899, III, No. 13; Centralbl. f. allg. Path. u. pathol. Anat., July, 18, 1900.

[4] La settim. med., Nos. 29 and 30, 1899; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[5] Centralbl. f. allg. Path. u. pathol. Anat., June 15, 1900.

[6] La Clin. mod., No. 51, 1899; Centralbl. f. allg. Path. u. pathol. Anat., July 18, 1900.

[1] Beiträge zur pathol. Anat. u. zur allg. Path., Bd. XXVII, Heft 2, 1900.

# NERVOUS AND MENTAL DISEASES.

By ARCHIBALD CHURCH, M.D.,

OF CHICAGO.

## SYMPTOMATOLOGY AND SYMPTOMATIC DISORDERS.

**Epidemic Intercostal Neuralgia.**—T. F. Riley [1] reports an epidemic marked by a feeling of distention in the right loin, as if due to a swollen spleen, attended by fever, nausea, and intercostal pain, and frequently the development of zoster near the splenic region or at a distance, sometimes on the face. Although the pains and herpetic manifestations came on in some instances in paroxysms, neither the examination of the blood nor the use of quinin gave any weight to the idea of malaria. The author also calls attention to an article in the Medicinische Wochenschrift for September, 1899, in which Wille reports over 100 cases occurring in the limited area of a small German city. [The probability of grippe causing these epidemic manifestations is not considered by these authors, though grippe in recent years has been particularly marked by neuralgic symptoms.]

**Enuresis.**—Amat,[2] limiting his consideration to what he calls the true neurotic type of enuresis, where there is no objective cause, such as phimosis, calculus, etc., considers that there are several varieties demanding distinct treatment. In his first variety the vesical muscular fibers are too sensitive to distention. During the day they are mastered by the will to some extent, but contract as the patient drops off to sleep. For this variety bromids, chloral, opium, and, better still, belladonna, atropin, and antipyrin, are indicated. In the second variety there is hyperesthesia of the vesical and urethral mucous membrane even when the bladder is flaccid. The condition is revealed by the catheter, and is met or cured by bromids, valerian, asafetida, and ammoniosulphate of copper. In the third variety the ease with which the catheter can be passed through the membranous part of the urethra shows that the vesical sphincter is not irritable, but weak. Here strychnin, rhus aromaticus, and, above all, electricity, are needed. Fourth, there seems to be a form of enuresis of a purely mental character, in which the child must be aroused and made to micturate 2 or 3 times a night. Physical exercise and general treatment here play the major part.

**The Sleeping Sickness.**—F. W. Mott [3] makes an additional report upon the 2 cases of sleeping sickness mentioned in the YEAR-

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[1] Med. Rec., Nov. 5, 1899.

[2] Bull. Gén. de Thérap., Nov. 8, 1899.

[3] Brit. Med. Jour., Dec. 16, 1899.

Book for 1899. Both cases terminated fatally and the microscopic appearances were essentially similar. In both, leptomeningitis and encephalomyelitis were found. Throughout the whole central nervous system, and especially in the medulla and at the base of the brain, the pia-arachnoid was infiltrated with mononuclear leukocytes, and the inflammation could be traced along the blood-vessels and septa into the nervous system. Both the clinical history and the morbid processes point to the chronic processes. It might, therefore, be attributed to a parasite, but to the author's mind, the evidence is insufficient, and there are some clinical features that resemble pellagra, so that the disease may be due to bad food, as is believed by some writers. Possibly it is due to an infectious organism for which a suitable stain has not yet been found. The author points out that the poison, whatever it may be, acts especially upon the lymphatic system, and particularly upon that of the central nervous system. The bacillus isolated by Gagigal and Lepierre was not found, although a careful search was made. The case reported by Stephen Mackenzie a few years ago and the microscopic sections derived from that case correspond entirely to the findings by the author.

**Cerebral Ataxia.**—Sanger Brown [1] reports 3 cases of persistent ataxia associated with cerebral disease, and refers to a number of other instances in the literature. These cases he describes under the title of "permanent nonprogressive ataxia," and in two instances they seemed secondary to some infectious process. [See also Year-Book for 1900, article "Cerebral Ataxia."]

**Babinski's Sign.**—F. W. Langdon [2] gives a very satisfactory outline of what is known of this sign, especially a report made upon it by Collier, whose conclusions were published in the Year-Book for 1900. He tabulates 110 cases in which the sign was present in conjunction with disturbance of the pyramidal tracts.

G. L. Walton and W. E. Paul [3] contribute a very important study of this valuable sign. They found Babinski's reflex in 70% of hemiplegics and diplegics, and in the same percentage of other cases with disease involving the pyramidal tract in the spinal cord. They never found this sign in health, and they doubt its existence in either functional or organic nervous or other disease not implicating the pyramidal tract. They consider that this sign is one of the earliest to appear in disease of the pyramidal pathway; for instance, at the onset of a hemiplegic attack, before exaggeration of the knee-jerk and ankle-clonus appear; and it may persist when other reflexes are absent, as, for example, when the knee-jerk and ankle-clonus are absent on account of ankylosis, contracture, and muscular wasting. This sign occasionally appears in meningitis, hydrocephalus, poisoning by alcohol or in uremia—those conditions, in other words, which furnish a disturbance of the cortex and of the upper end of the pyramidal path. Their conclusions, which are based upon the investigation of a very large number of patients, seem to have been reached with the greatest care, and are substantiated by the results obtained by other observers. This sign is one of extreme importance and value in connection with injuries and disorders of the pyramidal tract.

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[1] Am. Jour. Med. Sci., June, 1900.

[2] Cincin. Lancet-Clinic, Feb., 1900.

[3] Jour. Nerv. and Ment. Dis., June, 1900.

**Kernig's Sign.**—F. A. Packard [1] reports 3 cases of verified meningitis in infants, 2 being 16 months and 1 being 4 months old, in which Kernig's sign could not be elicited. He believes the sign to be of less value in the early years of life than later.

**Convulsions.**—Muehlig [2] reports, in a man 23 years of age, typhoid marked by epileptiform convulsions. On the twentieth day of the convalescence clonic spasms began in the outer fingers of the left hand, passed on to the eyelid, and then became general, lasting half an hour. The pupils were dilated and stationary. Another similar attack occurred 3 hours later, lasting 20 minutes; and after 5 hours, one lasting 10 minutes; 6 hours later a final attack, with interruptions, of an hour's duration. Between the attacks the patient felt well, but subsequently complained for 10 days of numbness and formication in the two outer fingers of the left hand. The exact nature of the cortical disturbance is a matter of speculation. During the course of the typhoid the patient had been very somnolent, and at times delirious. [Typhoid is recognized as a cause of epilepsy by all competent writers, and Eberth's bacillus has been found in the meninges frequently enough to bring these two facts into probable relation of cause and effect.]

**Mirror-writing.**—Arthur Sweeney [3] contributes a very interesting paper on this subject, with the details of a number of cases. He believes that mirror-writing is almost invariably due to visual defects, and quotes Hotz and others, who were able immediately to correct the mirror-writing by supplying the patient with proper glasses. [This does not account for the cases of mirror-writing following hemiplegia, in which it is probably due to the easier centrifugal movements, those of writing away from the body, although the author is not inclined to accept this explanation.]

**Nodding Spasm.**—John Thompson [4] contributes a very interesting article on this neurotic manifestation of childhood, and in discussing its causation says that there is, first, a special age—4 to 12 months—during which the coordination and the movements of the eyes, with those of the head, are being provided. Second, there is the nervous element: the disorder occurs in nervous families, and during the weakness of convalescence from various diseases, and nearly always during the excitable period of teething. Third, the most probable explanation of the striking association of these cases with insufficient light is the eye-strain consequent thereto. He has noticed that almost invariably children so affected have been housed in dark rooms and under unfavorable conditions of illumination. Fourth, there is also at times a history of a blow on the head and of fright. Lastly, the presence of rickets is an important predisposing cause.

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[1] Arch. Ped., April, 1900.

[2] Münch. med. Woch., 1900.

[3] St. Paul Med. Jour., 1900.

[4] Festschrift in honor of Abram Jacobi, New York, 1900.

**Lumbar Puncture to Produce Anesthesia.**—Severeanu [1] reported 70 cases to the Thirteenth International Congress at Paris, in which he had produced anesthesia for operative purposes in the lower extremities by injecting hydrochlorate of cocain into the spinal canal. In all these cases he observed a general weakness, lasting 2 days, and sometimes causing great anxiety. Frequently vomiting was severe, with headache. An inconvenience arising from the procedure is furnished by the patient watching the operation, thereby producing sometimes mental shock of serious degree. In the same discussion Tuffier said that he had performed 125 operations—of which 58 were laparotomies—under cocain anesthesia by Bier's method, 5 of which died. In 4 instances he did not hold the anesthesia responsible, and even in the fifth case postmortem showed heart lesions and pathologic complications of the lung. Racoviceanu-Pitesci, of Bucharest, had also operated on 125 patients under Bier's method of anesthesia, using doses of from 1 to 4 cg. of the hydrochlorate of cocain. In 3 patients symptoms resulted of such a character as to necessitate artificial respiration and subcutaneous injections of ether, and he claimed to know of 2 cases in which death had followed the method. [This method properly should be credited to Corning, of New York. It is attended by considerable danger, and the limitations of its usefulness are not clearly defined as yet.]

**Nervous Complications of Diabetes Mellitus.**—Desbonnets [2] divides the nervous complications of diabetes into motor, trophic, sensory, and mental. Of the motor disorders, he enumerates muscular feebleness, often of sudden appearance in the midst of apparent health, and chiefly affecting the lower extremities. It tends to disappear with the reduction of sugar. Paralysis of various forms—namely, monoplegia, hemiplegia, and paraplegia—may occur. The cases of paraplegia being really due to peripheral neuritis with insidious onset, many of the symptoms grow worse at night. This form of motor disorder may also involve the upper extremities; it bears considerable resemblance to alcoholic peripheral paralysis, and sometimes is mistaken for tabes, owing to the diminished or abolished knee-jerk, especially as gastric crises, perforating ulcer, and anesthesia may be encountered. The sensory manifestations of diabetes consist in neuralgia, especially involving the sciatic, the trifacial, the intercostal, and the cervico-occipital nerves. These various regions may be coincidentally affected, or the condition may be bilateral, and such combinations give assistance to the diagnosis. Gastralgia, arthralgia, and angina pectoris are encountered, and the special senses may be affected. Taste and smell may be lost or perverted. Deafness is also encountered. Amblyopia and ocular paralysis also occur. Amblyopia may be temporary or permanent. Among the trophic lesions, perforating ulcer, muscular atrophy, loss of nails and hair, and involvement of the nail-matrix are enumerated. The mental symptoms vary from profound depression with delusions to simple disturbance of memory and affections. Narcolepsy, mania, and coma are well known. All these nervous symptoms, according to the author, have little prognostic significance, as they appear in many different degrees in different patients at various stages of the disorder.

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[1] Brit. Med. Jour., Aug. 25, 1900.

[2] Thèse de Paris, 1900.

**Apomorphin as a Hypnotic.**—Douglas [1] calls particular attention to the use of apomorphin as a hypnotic, used hypodermically in doses that produce no nausea. About $\frac{1}{30}$ of a grain, more or less, adjusted according to the requirements, he finds quite harmless, and perfectly competent in mild insomnia and in furious delirium, producing sleep within 25 minutes. No disagreeable after-effects were noted and no possibility of acquiring a drug habit exists, as an increase of the dose leads to violent vomiting. He has used the remedy in 300 cases, failing of good effect in only 2 or 3 instances, in which, moreover, large doses produced no emetic effect. [In restlessness not due to pain or mental disturbance apomorphin in the foregoing dose acts well.]

**Adiposis Dolorosa.**—F. X. Dercum [2] returns to this subject, having had an opportunity for autopsic investigation in one of the cases formerly reported by him in various journals. Microscopically the fatty tissue presented nothing to distinguish it from ordinary fat, but the peripheral nerves found in it showed undoubted interstitial neuritis. The spinal cord presented slight degeneration of the columns of Goll in the cervical and upper thoracic regions. The thyroid gland was made up of 3 or 4 different kinds of secreting tissue, modifications of a normal state. In two other cases of this affection the thyroid was also apparently involved, though never microscopically examined.

Charles W. Burr [3] also reports a case of adiposis dolorosa with autopsy. Clinically the case conformed closely to those described by Dercum. The patient finally died, after a prolonged somnolent and comatose state, from uremia due to nephritis. In the right lobe of the thyroid gland there was a spheric concretion as large as a hickory-nut. On microscopic examination of the thyroid colloid degeneration and absence of secreting cells in many acini were found. There were also many areas of small round-celled infiltration and indications of active inflammatory processes. The muscles showed marked degeneration by the osmic acid method, probably secondary to degeneration of the nerves, as a medium grade of interstitial neuritis of the intramuscular nerve-bundles was present. There was acute parenchymatous nephritis. The ovaries were also diseased; they were sclerosed and not performing their great function. In this case, as well as in that of Dercum, there was disease of the thyroid gland and of the finer nerve-branches. The condition of the ovaries is suggestive, as after castration there is a tendency to obesity. The author concludes that at present we can only say that "adiposis dolorosa is a clinical entity, having definite signs and symptoms, but with as yet no known pathology."

W. H. White [4] reports a case of adiposis dolorosa, and the illustrations strongly recall the instances published by Dercum. The case

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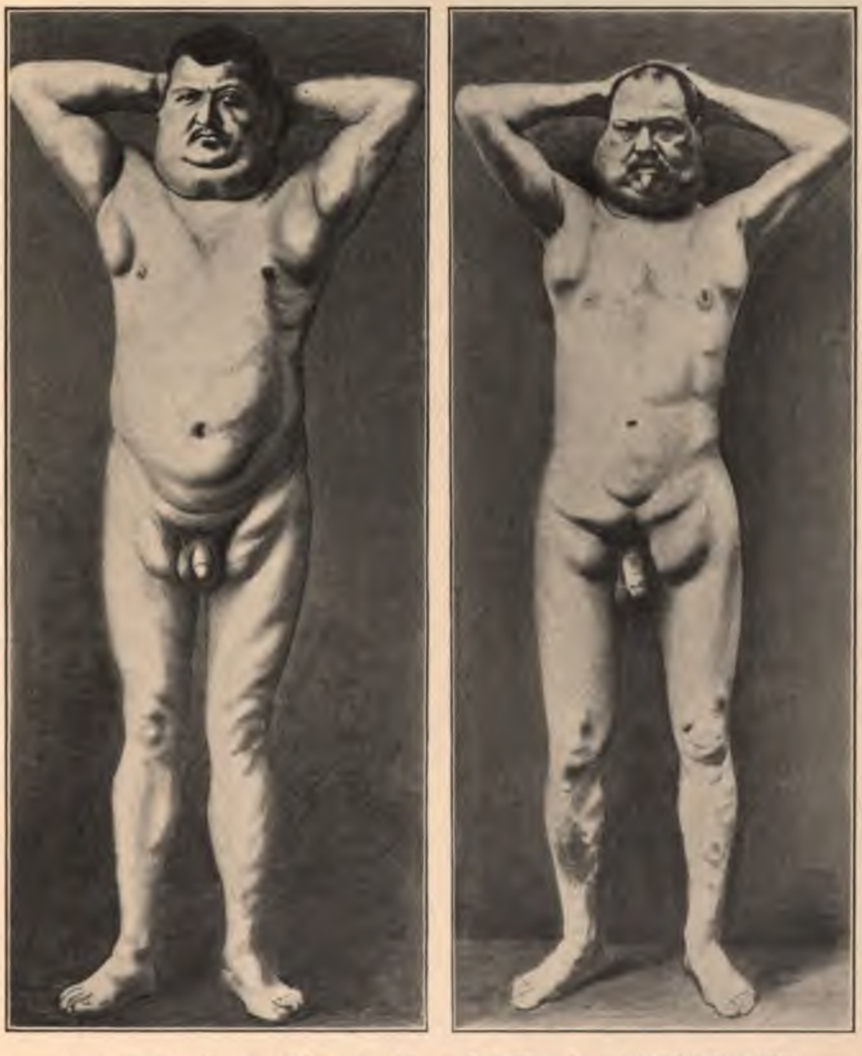
[1] Merck's Archiv, June, 1900.

[2] Jour. Nerv. and Ment. Dis., Aug., 1900.

[3] Jour. Nerv. and Ment. Dis., Oct., 1900.

[4] Brit. Med. Jour., Dec. 2, 1899.

PLATE 4.



Symmetric adenolipomatosis (Launois and Bensaude, in Nouv. Icon. de la Salpêtrière, 1900).

reported differs from the majority in the literature in having appeared early in life and with painful symptoms more marked in the early stage than lipomatosis. Recurrent attacks of mental disorder were presented, but the patient did not give the usual history of syphilis, alcoholism, and the indications of neuritis, though there was a suggestion of some toxic factor.

**Symmetric Adenolipomatosis.**—Launois and Bensaude[1] under this title make an extensive contribution to the subject of multiple symmetric fatty deposits unattended by general symptoms of illness or physical disorder, though they have noticed some exceptions to this rule. For instance, a general feeling of weakness or of fatigue is reported in numerous observations, and in some cases irritability, apathy, and even hypochondriasis. These local masses of fatty tissue, as the authors well point out, seem to enjoy an individuality, preserving their volume during conditions under which the patients generally become cachectic, and even in the presence of considerable emaciation secondary to tuberculosis, Bright's disease, etc. As a rule, they are without painful symptoms or sensory disturbances, though in this connection the analogous or identical adiposis dolorosa described in this country should perhaps be borne in mind. The authors find about 80 cases described in the literature. It is much more common among men than among women. In women the neck is more frequently spared, the lipomatosis having a tendency to invade the shoulders and hips. It usually makes its appearance after 20 years of age; the youngest case was 21 and the oldest 58. Alcoholism was found in 30 % of the cases. The disease often coincides with syphilis, albuminuria, cancer, gout, asthma, and varicose conditions. The authors do not believe, contrary to the statement of Madelung, who first in Germany gave prominence to the disorder, that the affection never coincides with obesity. Generally, however, obesity is not marked. They adduce numerous points of resemblance between the condition of adenolymphocele and adenolipomatosis, and make the following arguments: (1) That the fatty infiltration penetrates along the lines of the lymphatic vessels. (2) That the aptitude of these tumefactions to increase and decrease with astonishing rapidity can be explained only by intimate connection with the circulatory system. (3) That the lymphatic theory explains much better than the nervous theory all the facts that have been accumulated in relation to the disorder. It explains the general localization of the tumefactions, their symmetry, their frequency, and especially their constancy in the neighborhood of the neck, axillas, and groins. They would denominate it, at least for the time being, a disease of the lymphatic system.

## DISEASES OF CEREBRAL MENINGES AND CRANIAL NERVES.

**Trifacial Neuralgia.**—W. H. Bennett[2] reports 9 cases of trigeminal neuralgia treated by the injection of osmic acid into the substance of the nerve. His plan appears to have been to uncover the nerve at some point where it is subcutaneous and then to inject into the nerve-trunk from 5 to 10 minims of a 1.5% solution of osmic acid. In every instance the pain was immediately controlled, and spasm of the muscles, when associated with pain, also subsided at once. Recurrence took place in one case after some months, and was again relieved by the same simple expedient. Sufficient time, however, has not elapsed in any instance to justify a statement as to ultimate results.

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[1] Nouv. Icon. de la Salpêt., 1900.

[2] Lancet, Nov. 4, 1899.

L. F. Barker,[1] after giving a detailed report of several Gasserian ganglions removed in neuralgic conditions, concludes: (1) If a ganglion be entirely removed, there need be no fear of a return of pain from irritation of the stump of the nervus trigeminus left behind, for all the axons of this stump will degenerate to their terminations in the pons and medulla, down as far as the cervical cord. The end of a nerve in an amputation stump is not analogous. (2) Complete removal of a Gasserian ganglion utterly abolishes the possibility of calling forth sensations in consciousness by applying stimuli to the domain of peripheral distribution of the nerves connected with the ganglion of the corresponding side; retention of sensation on peripheral stimulation after operation indicates incomplete removal. (3) If pain persists, paroxysmally or continuously, after complete removal of the ganglion, or after evulsion of the nervus trigeminus from the pons, the ganglion being left *in situ*, a lesion of the central neurons—of the second or of the higher orders—of the trigeminal afferent conduction path is indicated. (4) In tic douloureux due to disease of the peripheral set of trigeminal sensory neurons relief should be as complete and permanent by cutting the nervus trigeminus between the ganglion and the pons, and evulsing the central end without removal of the ganglion, as when the ganglion itself is excised.

Articles [2] on this subject by a number of American authors include one by Robert Abbé, of New York, who describes modifications of older methods of operation, which promise better results. Charles L. Dana, in discussing the natural history and treatment of the disorder, says: "I should say that the early forms of tic douloureux, such as I have called a 'migrainous tic,' occurring usually in women, should not be operated on. There are some exceptions to this, however, in which tic douloureux occurs in early life, due to a distinct local disease, such as inflammation of the nerve, or of the antrum, or of the jaws. In true tic of the degenerative period of life prompt medicinal treatment will usually control the disease, and operation is rarely indicated at first. In tic which has lasted three or more years it may be safely said to the patient that medicinal treatment may produce a remission, and that this remission may be repeated, and that eventually the disease may be controlled by repeated treatments, but this is not at all sure. It may be said here, too, that a minor operation may give more relief than medicinal treatment. The question of prescribing major operations must be decided in each individual case, on its special merits."

F. Krause, of Berlin, in the Section of Surgery at the recent International Congress of Medicine, detailed 24 cases of intracranial resection of the trigeminal nerve. In none had there been any recurrence of pain, and all had been operated according to the Krause method. The cases operated on had now been observed for a period of from 3 to 7½ years, and their ages at the time of operation ranged from 30 to 72. Two cases died, one directly, from operation by collapse; and the other 6 days after operation, from sclerosis of the coronary arteries and cardiac insufficiency, the wound being completely healed.

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[1] Jour. Am. Med. Assoc., May 5, 1900.

[2] Jour. Am. Med. Assoc., May 5, 1900.

**Lesions of the Optic Chiasm.**—W. M. Leszynsky[1] reports 3 cases of lesions of the optic chiasm, briefly reviews the literature, and states that chiasm lesions may be divided into 4 classes: (1) Associated with intracranial growths and their concomitant symptomatology; (2) from enlargement of the prehypophysis cerebri, as occurring in acromegaly; (3) in syphilitic basal meningitis; (4) from a circumscribed pathologic process, which gradually produces complete atrophy of both optic nerves, without any cerebral symptoms whatever.

**Meningitis.**—Nobecourt and Delsarte[2] report a case of so-called acute serous meningitis which at autopsy, in addition to the lesions of bronchopneumonia, showed a congested condition of the meninges at the convexity of the hemispheres, more marked posteriorly. The cerebrospinal fluid was clear and limpid, as was also that of the lateral ventricles. No solid exudate and no tubercles were present. Cultures of the cerebrospinal fluid obtained during life, and also at autopsy, showed the presence of the pure streptococcus. In another case presenting acute meningitic symptoms for 2 days before death the autopsy revealed the lesions of a seropurulent cerebrospinal meningitis, and here bacteriologic examination of the spinal fluid and of that of the ventricles showed the presence of the streptococcus, analogous to the one observed in the first case. The same organism, therefore, determined the presence of a serous inflammation in the one case, and in the other of a seropurulent one, which the authors are inclined to attribute to a peculiar predisposition or special power of resistance in the two instances, but acknowledge that these factors are indefinite and not well understood. The organism presented peculiarities. Upon jelose it grew poorly, giving minute colonies resembling pneumococcus. After several generations it acquired the typical characteristics of the streptococcus, giving in liquid cultures a deposit made up of clumps which had the form of the diplococcus at first, and later presented long chains when cultivated in the blood-serum of the rabbit, and possessed no capsule taking stain. It was harmless for rabbits and mice.

R. B. H. Gradwohl[3] reports the transmission of cerebrospinal meningitis from mother to fetus. The mother died of the disease, and at the autopsy the usual findings of cerebrospinal meningitis were made. A 7-months' fetus was removed from the uterus and presented the exact counterpart of the condition of the maternal meninges, with perhaps more of a seropurulent than a purely purulent exudation. Diplococcus intracellularis meningitidis was found in the meninges of mother and child. The case was one of an epidemic of 34 cases previously described by the author, who mentions that Herwerden also reports a case of sporadic meningitis in mother and fetus.

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[1] Jour. Nerv. and Ment. Dis., Mar., 1900.
[2] Ann. de méd. et chir. Infantiles, April 15, 1900.
[3] Phila. Med. Jour., Sept. 2, 1899.

E. P. Joslin [1] contributes an article on **hydrocephalus secondary to cerebral meningitis**, and reports 8 cases which presented considerable uniformity. In all save one the hydrocephalic fluid was clear and colorless, and in 2 instances it was sterile. In a third case diplococci were found, though not the diplococcus of Weichselbaum. The sterility of the fluid is explained by the fact that bacteria regularly disappear from the brain in the early part of the disease. In all cases the pia was thickened in the region of the roof of the fourth ventricle, and in most cases at the base of the brain and elsewhere. This thickening, which is dense and tough, is due to the formation of granulation tissue starting from the pia and incorporated with the fibrinous exudation, which it gradually removes. It covers the surface, not only barring the foramen of Magendie and the lateral openings, but rendering the roof of the ventricle impervious. The changes in the brain resulting from pressure are those ordinarily found in hydrocephalus. The flattening of the convolutions amounted to one-third of their dimensions. The endyma in the first case was swollen and velvety, somewhat thickened in the second, and smooth in the third; very slightly granular in the fourth, fifth, and sixth cases. Nothing abnormal was observed in the seventh and eighth. The condition of acute hydrocephalus seems to depend upon the plastering of the meninges at the base of the brain and in the region of the fourth ventricle, blocking the foramina and rendering the velum interpositum impervious. There seems to be no relation between the development of hydrocephalus and the severity of the original attack, leading to the supposition that the location of the inflammation is the essential feature. The especial symptoms seem to be apathy and mental lassitude. Vomiting took place in all but one case. Headache was severe in half the cases, and in 3 of the other cases it was but of a mild type. The pupils were dilated or reacted sluggishly or not at all in 7 of the patients. Muscular weakness and emaciation were the rule. The temperature curves were suggestive; they were normal or subnormal during much of the illness, especially for a few weeks before death. The relation of temperature to pulse is striking, the pulse rapidly rising as the temperature falls, and vice versa. Toward the end the pulse falls, and then there appears from hour to hour great irregularity in the rate, but this is a terminal manifestation often associated with Cheyne-Stokes respiration. The duration of the various cases was 89, 52, 90, 60, 40, 82, and 131 days.

**Centers for the Pupils in Accommodation.**—Von Bechterew [2] contends that on the anterior margins of the occipital lobe there is a center stimulation of which produces narrowing of the pupil and increased accommodative effort. In his laboratory, under the

[1] Am. Jour. Med. Sci., Oct., 1900.

[2] Neurol. Centralbl., May 1, 1900.

observation of Belitzki, this has been proved by experiments upon dogs. The action of the accommodative apparatus was demonstrated by introducing a needle into the ocular lens, every modification of its shape being indicated by the movements of the end of the needle. Stimulation of the region in question gave rise to such movements as could easily be understood. Roux,[1] it will be recalled, asserts that in this region there is a full representation of the oculomotor apparatus with particular representation of reflex coordinating movements.

**Asthma.**—W. A. Wells [2] contributes an article on the subject of asthma, in which he concludes in part as follows: (1) Asthma has so many points of resemblance with migraine, angina pectoris, and epilepsy that the suspicion may reasonably be entertained that they all have a similar pathology; (2) of the theories which have been advanced to explain the pathogenesis of the asthmatic paroxysm, no one of them so well harmonizes all the facts as the vasomotor theory; and this only when we admit that the disturbance is essentially only that of arterial contraction, rather than dilation; (3) there is always present in asthma a morbid constitutional state, affecting especially the sympathetic nervous system and giving rise to a certain nutritional aberration, whose most salient feature is the increase of uric acid and the urates; (4) asthma occurs as a reflex neurosis from diseases of different organs, but especially often from those directly supplied by branches of the vagus nerve; the nasal trouble, which is the most frequent reflex cause, is not necessarily an obstruction, and may be very inconspicuous and difficult to detect; (5) nearly all cases of asthma show evidences of a pronounced psychic element, as in the curious variety of exciting causes of the attack, in the capriciousness of its course and behavior, and its dependence upon emotional states, suggestions, etc.; (6) in the treatment of the paroxysm of asthma a strict individualization needs to be observed; the best remedies are those which overcome arterial spasm, such as morphin, nitroglycerin, atropin, and chloral hydrate; (7) treatment in the intervals of the attacks must be directed, in the first place, to the removal of the cause, and, secondly, to the institution of sound hygienic and prophylactic measures in relation to the mind, diet, air, exercise, etc.; (8) medicinal treatment is addressed to improving the constitutional state, for which the best remedies are piperazin, potassium iodid, and the other iodids, the alkalies, and general tonic treatment.

## DISEASES OF BRAIN PROPER.

**Cerebritis.**—Charles L. Dana [3] reports an interesting case of hemorrhagic encephalitis in a man of 67 who had been an excessive, steady drinker, and who also presented malaria, in addition to which he had several years previously suffered from a severe freezing and a sunstroke. A number of hemorrhagic inflammatory foci were found in the brain, medulla, and spinal cord, presenting the large epithelioid cells commonly encountered in this disorder, but peculiarly limited to the white matter in this instance. The author outlines 3 varieties of nonsuppurative encephalitis: First, the infectious form occurring in infants, originally outlined by Struempel, but not, as supposed by him, confined to the cortex. Second, hemorrhagic polioencephalitis involving the gray matter of the floor of the fourth ventricle and the aqueduct. Third, the hemorrhagic encephalitis of adults, almost invariably secondary to influenza, and sometimes called influenzal encephalitis. The case detailed does not correspond to this third type in clinically lacking the history of influenza, but the author is disposed to consider malaria an equivalent.

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[1] YEAR-BOOK, 1900.

[2] N. Y. Med. Jour., Oct. 20, 1900.

[3] Med. Rec., July 7, 1900.

**The Influence of Cranial Traumatism in the Production and Evolution of Diseases of the Brain.**—At the Thirteenth International Congress of Medicine, Section of Neurology, Ernst Ehrnrooth,[1] of Finland, reported experiments made upon about 130 rabbits, and found that traumatism of the head predisposes, upon the intravenous injection of a culture of streptococcus or staphylococcus, to microbic infection of the brain or the meninges. The infection is most frequent at the site of violent blows and develops most commonly in the portion of the head exposed to the traumatism, and with a greater intensity than on the opposite side of the head. In these experiments 76.5 % of the animals struck and immediately infected, and 56 % of the animals struck and infected at a later date, presented a microbic infiltration of the brain; while in control animals merely infected, only 13.5 % showed involvement of the brain. He believes that his experiments give support to the opinion that traumatism of the head acts as a predisposing cause in the development of infectious diseases of the brain, even in the absence of any disease of the neighboring sensory cavities, the pyogenic agents reaching the brain indirectly by way of the blood.

**Brain Tumor.**—G. W. McCaskey[2] reports a case of cerebellar tumor in which there was drainage of fluid through the nose. About 2 months before death the patient began to have through the right nostril a free discharge of serous fluid, which ran as a steady stream or dropped rapidly, and amounted to several ounces daily. At the time this discharge began there had been absolute deafness for about 3 months, but within an hour her hearing partly returned in the right ear, and so remained until her death. The base of the skull, against which the tumor pressed, was found to be eroded down to the cancellous structure. Another point of interest in the case is the fact that optic neuritis was decidedly greater on the side corresponding to the tumor. He has met the same condition in a second case.

G. Biancone[3] makes a comprehensive study of the subject of **tumors of the corpora quadrigemina,** adding the details of a carefully observed case, taken with other cases in the literature—19 all told. The symptomatology may be briefly stated as follows: (1) Disorders of vision are generally present; (2) paralysis of the external eye muscles is very frequent, is usually bilateral, and develops gradually; (3) pupillary disturbances are frequent—inequalities, dilation, contraction, Argyll Robertson phenomenon, etc.; (4) ataxia is common; (5) disturbances of hearing—as deafness, hyperacusia, and tinnitus—commonly appear late; (6) paresis, especially of hemiplegic form, often complicates the picture; (7) contracture, spasms, and convulsions are rare; (8) disorders of sensation are rare; (9) headache and vomiting occur only upon the increase of intracranial pressure; (10) mental disturbance, disorders of speech, vertigo, and nystagmus have been occasionally noticed; (11) the condition of the reflexes is variable; (12) slowness of the pulse and loss of muscular sensation have been noticed. The author calls attention to a particular symptom, a sort of priapism with considerable increase of the genesic sense, persisting from the beginning of the sickness in his patient and not formerly noted, which corresponds to what is observed in vertebrates after the ablation of the quadrate tubercles.

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[1] Rev. Neurol., Aug., 1900.

[2] N. Y. Med. Rec., Mar. 31, 1900.

[3] Rivista Sper., Dec., 1899.

R. T. Williamson [1] calls attention to the fact that **tumor and abscess in the prefrontal lobe** may abolish the knee-jerks, just as occurs in cerebellar tumor. The knee-jerks were lost in 3 out of 5 cases that have fallen under his observation, nothing else serving to explain their disappearance aside from the prefrontal tumor. A similar case is reported by Gordinier in the "American Journal of the Medical Sciences" for May, 1899, referred to in the YEAR-BOOK for 1900, where the tumor was situated in the second frontal convolution on the left side.

R. T. Williamson [2] reports 5 cases under his own observation and 45 in the literature in which gross changes in the prefrontal region, the result of tumor or abscess, presented a **loss of the patellar reflex** in 20%. He calls attention to the similarity in disease of the cerebellum. The patellar loss was bilateral.

Dupont [3] has used injections of tuberculin to establish a diagnosis of **tuberculous brain tumor.** He reported two observations. A patient with brain tumor and a syphilitic history furnished the characteristic reaction of tuberculous lesions. The other patient, also syphilitic, did not react. [The suggestion is perhaps valuable, although in neither case was the exact character of the tumor verified.]

F. E. Batten and James S. Collier,[4] from a study of **changes of the spinal cord associated with cerebral tumor,** conclude: (1) That degeneration of the posterior columns of the spinal cord frequently occurs in cases of intracranial tumor—*i. e.*, about 65%. (2) That such degeneration is more liable to affect the spinal cord in the cervical region than in the dorsal or lumbar regions, and is more marked in the postero-external than in the postero-internal columns. (3) That such degeneration is of root origin, and arises from the point where the root enters the spinal cord; the posterior roots are always less affected than the posterior columns, and may show no degeneration. (4) That such degeneration is due to traction on the posterior roots by the distention of the arachnoid, owing to the increased intracranial pressure, and especially such as tends to rapid dilation of the ventricles and the subarachnoid space in the spinal cord. (5) That such degeneration is independent both of the situation and of the nature of the tumor, except so far as their liability to give rise to the preceding condition is concerned. (6) That optic neuritis bears no relation to the occurrence of the posterior degeneration. (7) That absence of the knee-jerks, except in the semicomatose state, indicates posterior degeneration, but that their presence does not negative such a condition, and absence of the arm-jerks has the same significance. (8) That degeneration may occur in the direct cerebellar tract, such degeneration being directly due to pressure on the cervical region of the cord.

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[1] Glasgow Med. Jour., Nov., 1899.

[2] Glasgow Med. Jour., Nov., 1899.

[3] Thirteenth Internat. Cong., Sec. on Neurology; Rev. Neurol., Aug., 1900.

[4] Brain, 1899.

G. L. Walton and F. E. Cheney,[1] after investigating the subject of **tumors of the pituitary body,** report a case and give the literature. They reach the following conclusions: (1) Congenital peculiarities in growth, resembling those of acromegaly, but occurring in otherwise healthy individuals, may point to a structural defect of the pituitary gland—a defect sometimes furnishing the starting-point for new growth later in life. (2) The occurrence of pituitary tumor without definite symptoms of acromegaly does not necessarily disprove a connection between this organ and this disease, for the persistence of even a small amount of healthy gland tissue is sufficient fairly to carry on the function of the pituitary body. (3) The combination of general symptoms of new growth with optic atrophy and loss of temporal field of vision makes the diagnosis of pituitary tumor almost certain. (4) Hemiachromatopsia is not necessarily of central origin.

**Cerebral Diplegia.**—J. S. Collier [2] presents a critical résumé of the important subject of cerebral diplegia in childhood, introduced by a detailed account of several cases, with pathologic reports by Risien Russell. He summarizes as follows: One-third of the recorded cases of cerebral diplegia are without known clinical antecedents; maternal morbid states are probably by far the most important clinical antecedents, and such states have probably existed in many of the cases to which no definite cause can be assigned; premature birth has probably no causal relation with diplegia; the causal relation of birth injuries and asphyxia neonatorum to this condition has been much overestimated, and the absolute proof of it is as yet wanting, notwithstanding the frequency of clinical association; some cases certainly follow cerebral vascular lesions; acute diseases sometimes precede the onset of symptoms, but whether in all cases as the initial factor in disease, or in some cases only as an immediate exciting cause, has not been determined. With regard to the pathology of diplegia, he recites the following conclusions: (I) The cerebral lesions found in nearly all cases of diplegia are (1) atrophy of the convolutions; (2) porencephaly; (3) no coarse lesion, but degeneration and disappearance of the cortical neurons. (II) The initial morbid processes producing diffuse and lobar atrophic sclerosis are (1) degeneration of the neurons of the cerebral cortex; (2) vascular lesions, embolism, thrombosis, and intracerebral hemorrhage; (3) perhaps meningeal hemorrhage. (III) Porencephaly may be produced by (1) arrest of development, with tissue absorption, owing to fetal vascular disease; (2) direct injury to the brain; vascular lesions after birth; embolism, thrombosis, and intracerebral hemorrhage; (3) it may also result from the process of tissue absorption and shrinking of brain-substance occurring in cerebral sclerosis, and may result as a secondary process in degeneration of the cortical neurons. (IV) Bilateral spastic hemiplegia is connected with deep lesions of the hemispheres. The other forms of diplegia are connected with superficial lesions of the hemispheres. (V) Athetotic and choreic diplegia are associated with superficial lesions of the cortex. In these types lesions of the corpora striata are not constant. The author would consider that the degenerative initial lesions are the result of the toxic process, a position strongly supported by Jendrassik and Marié. But in familial nervous diseases it is natural to look to the neuron as the probable seat of the initial disease, even where it is possible to conceive the process secondary to some toxic state.

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[1] Boston M. and S. Jour., Dec., 1899.

[2] Brain, Autumn, 1899.

W. Koenig [1] contends that many cases of diplegia in childhood are **due to syphilis,** and reports 3 instances, and finally 2 others, in which the luetic factor could be incriminated.

**Hemiplegia.**—Sicard [2] reports clinical observations made on the subject of the abdominal muscles in this condition. Hitherto it has been supposed that the abdominal muscles were spared. Twenty-two patients suffering from hemiplegia of organic origin were examined by careful and repeated palpation during ordinary respiration, during the act of coughing, and during deep breathing, and particular attention was paid to the condition of the inguinal orifices. As a result, it appeared that a distinct paresis of the abdominal muscles could be obtained on the hemiplegic side.

**Cerebral Syphilis.**—The involvement of single functions of the oculomotorius has long been considered diagnostically valuable as indicating a nuclear lesion. Wilbrand and Saenger [3] give a table of 14 cases in which the lesion was situated elsewhere than in the nucleus, being orbital, basilar, or cortical.

**Multiple Sclerosis.**—R. Balint [4] reports a case of multiple sclerosis with postmortem section and histologic study. He concludes that we must reach the conclusion that multiple sclerosis is not a disease entity, but merely a symptom-complex based upon various pathologic processes.

**Huntingdon's Chorea.**—C. A. Good [5] reports a case of this disease, embracing a careful examination of the entire nervous system. No evidence of any inflammatory process was found, but, on the other hand, there existed throughout the cortex involvement of the ganglion cells, most marked in the motor and frontal regions. This was degenerative in character, attended in its later progress by fatty changes and the presence of fat granules. Several excellent illustrations are furnished.

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[1] Neurol. Centralbl., April, 1900.

[2] Arch. de neurol., Dec., 1899.

[3] Neurol. des Auges, 1900.

[4] Deut. Zeit. f. Nervenh., 1900.

[5] Am. Jour. Insan., July, 1900.

Ladame[1] examines **the psychic state in hereditary chorea.** Irritability of character is one of the essential traits, but he denies that suicide, asserted by Huntingdon to be common in this disease, is so in fact. In the literature he is unable to find a single case of suicide or attempt at suicide. Melancholia at first is not so constant as is usually supposed, but all forms of psychoses are observed; yet hallucinations, delirious ideas, and paranoiac manifestations are accessory symptoms of the affection, while ideas of grandeur or persecution assume the characteristics of those common in mental debility and not those of paranoia. Progressive enfeeblement of the mental faculties is alone the essential characteristic, though psychic signs are a constant phenomenon in the disease.

**Neurasthenic Bulbar Paralysis.**—Thomas Buzzard,[2] under the title "myasthenia gravis pseudoparalytica," publishes a clinical lecture on the subject. The general muscular system is involved, and special importance is attached to the myasthenic electric reaction. The muscles respond to faradism in varying degrees on different occasions, the tendency being for the response to disappear if the muscle has been fatigued by voluntary exertion, and some muscles tire very much more easily than others. Also, as a result of the faradic stimulation, voluntary power in the muscle is diminished and a feeling of fatigue is induced. As to galvanism, after repeated stimulation contractions decrease, but the muscle can not be tired to the same extent as is the case with the faradic current. There are no polar changes. The behavior of the knee-jerks in these cases is interesting. Repeated tapping on the patellar tendon failed to produce any diminution in the vigor of the response, and when the vastus internus had been completely exhausted by the faradic current, a vigorous knee-jerk immediately occurred upon tapping the tendon. The author thinks that there may be various conditions not separated by strongly defined lesions giving rise to this pseudoparalysis. As regards treatment, he states that little or nothing is to be said; one patient improved on thyroid extract, which had no effect at all upon another. [The whole subject is exhaustively reviewed in a monograph by Oppenheim entitled "Myasthenische Paralyse," Berlin, 1901.]

## DISEASES OF SPINAL MENINGES AND SPINAL NERVES.

**Multiple Neuritis.**—Sacquepee and Dopter[3] review the histories of cases of multiple neuritis **following malaria,** and report 3 of their own, one of which, however, was so complicated as scarcely with propriety to appear under that head.

F. J. Woollacott[4] tabulates a large number of cases of diphtheria, several thousand all told, with reference to the occurrence of **paralysis in diphtheria** as modified by antitoxin, and concludes that, on the whole, the percentage of paralysis is increased. There is some evidence that large doses, not less than 4000 units, are more effective than small ones in affecting paralysis and diminishing the mortality due to it; that the earlier the antitoxin is given, the less likely is paralysis to follow, and when it occurs after early injection, it is milder and of shorter duration; that the type of paralysis, on the whole, has become less severe, and that it is more prone to attack the young. The principal conclusion, the author states, is that the full value of antitoxin is obtained only by using early and sufficient doses. [It would seem from a study of the article that in a majority of the cases the paralysis is due to the fact that cases which would otherwise terminate fatally, under the influence of antitoxin are saved, but the paralytic features develop.]

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[1] Arch. de neurol., Feb., 1900.

[2] Brit. Med. Jour., Mar. 3, 1900.

[3] Rev. de méd., April, 1900.

[4] Lancet, Aug. 26, 1900.

E. E. Ware [1] reports a case of **involvement of the pharynx and palate** after a diphtheric infection in which the false membrane was located on the vulva. The case is particularly interesting as showing the vulnerability of the pharyngeal apparatus to the diphtheric poison, and disproves the contention that the palate and pharynx are usually most involved because of proximity to the common site of the false membrane.

R. Cestan [2] reports a case of **syphilitic multiple neuritis** occurring in the second stage of the disease, with slow recovery and a typical clinical picture. Multiple neuritis due to syphilis is of such unusual occurrence that by some it is doubted. The author is able to collect 13 cases from the literature, some of which he does not, however, accept as true to the caption. He is inclined to believe that it is of more frequent occurrence than is supposed because of its usually benign course.

Schuetz [3] reports a case of **erythromelalgia** with atrophy of the skin. The patient was a woman who had suffered much from malaria, acute articular rheumatism, and nervousness. The erythromelalgia affected the left arm and hand, with the usual symptoms of redness, severe burning pain, etc., especially after night. There was also headache, giddiness, buzzing in the head, and difficulty in hearing at times.

Keen and Spiller [4] report a typical case of **multiple neurofibromas** and make a study of the literature, from which they conclude, in brief, that one is justified in classing under a single head the neurofibromas of the nerves, the cutaneous fibromas, the plexiform neuromas, certain forms of elephantiasis, and certain pigment nevi, though they would not assume that all cutaneous fibromas arise in the nerves.

**Spinal Puncture.**—Alfred Hand [5] makes a study of the reports of various authors upon lumbar puncture. He notes the following bacteria as having been found in the cerebrospinal fluid: streptococci, pneumococci, meningococci, typhoid bacilli, Bacterium coli, tubercle bacilli. Blood has also been obtained by Jacobi in 2 cases of traumatism of the spine. The author concludes, first, that lumbar puncture has a wider field as a diagnostic than as a therapeutic means. Second, as an aid to diagnosis lumbar puncture is of value only when examination of the fluid gives positive results. Third, therapeutically it is of value in epidemic cerebrospinal meningitis in order to bring about recovery; in tuberculous meningitis, to promote comfort; and in other conditions of excessive pressure, to favor recovery by removing a condition immediately dangerous to life.

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[1] Lancet, 1900.
[2] Nouv. Icon. de la Salpêt., 1900.
[3] Derm. Zeit., Bd. VI.
[4] Am. Jour. Med. Sci., May, 1900.
[5] Am. Jour. Med. Sci., Oct., 1900.

**Meralgia.**—J. H. Musser and Joseph Sailer[1] report 10 cases of this disorder in the distribution of the external cutaneous nerve of the thigh, and make numerous reports of its literature. They state that the pathology is doubtful. Only one case has come to autopsy, and presented a spindle-shaped swelling in the nerve at the point where it crossed the crest of the ilium, and at this point there was evidence of neuritis and perineuritis of long standing. Bernhardt calls attention to the fact that in the majority of cases there is tenderness upon pressure at a point just below the anterior superior spine of the ilium. In intractable cases resection of the nerve may be recommended, and has been done by Souques. Complete relief was obtained, but anesthesia of course persisted in the distribution of the nerve, which histologically appeared normal, in that instance. The authors finally tabulate 99 cases, from which it appears that the disease is most frequent between the ages of 30 and 60. There were 75 males and 21 females, in 3 cases the sex not being mentioned. It was on the right side 32 times, 31 times on the left side, and 20 times bilateral, while in 16 cases the side was not mentioned. For further reference to this subject see previous YEAR-BOOKS.

E. Nawratzki[2] had the opportunity of examining the external cutaneous nerves of the thighs in a case of the sensory disturbance first described by Bernhardt and sometimes known as meralgia. Upon the removal of these nerves it was observed that there was a spindle-shaped swelling on each at the point where it passed over the brim of the pelvis under Poupart's ligament, and upon closer examination this was found to be a peri-interstitial and parenchymatous neuritis.

**Restoration of Movement after Nerve Section.**—Robert Kennedy[3] reports a number of experiments in which nerves were divided and sutured. Some were accurately placed in apposition, and in others the nerves were rotated on their long axes and then brought together so that the original pathways were decidedly displaced. In no case, upon subsequent examination, could continuity of nerve-fibers from the central to the peripheral segment be traced, and the author is doubtful whether restoration of function was by the old paths by decussation of nerve-fibers in the cicatrix. The practical conclusion is that in suturing a divided nerve no trouble need be taken to secure coaptation for the purpose of bringing proximal and distal fibers into exact relation, a simple approximation of the two ends being all that is required.

## DISEASES OF THE SPINAL CORD.

**Tabes.**—A. P. Francine[4] reports 2 cases of **tabes in negroes,** husband and wife, with antecedent syphilitic history. The contribution is made because of the rarity of tabes in the negro race and because of the association of the disease in man and wife. [During the past year 3 cases of tabes in the colored race have appeared in a single clinic. It is difficult to say that any one of the 3 individuals is without some admixture of white blood, although in one instance it seems to be entirely absent.]

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[1] Jour. Nerv. and Ment. Dis., Jan., 1900.
[2] Deut. Zeit f. Nervenh., 1900.
[3] Proc. Roy. Soc. Edinb., 1899.
[4] Am. Jour. Med. Sci., May, 1900.

H. Jullian [1] describes a case in which there was what he calls **nasal crises,** and thinks that it is the second case of the sort that has been described, the former one being published by Klippel. In both cases there was diminished sensibility about the nose and in the nasal mucous membrane. The crises were preceded by a curious sensation limited to the parts mentioned, and were followed by a feeling as if the nostril was stuffed up, with a prickly sensation around the larynx and finally a violent fit of sneezing. In the second case the principal manifestation was the sneezing, which consisted in a prolonged bout.

Fraenkel [2] contributes a very important paper on the **tendon reflexes in locomotor ataxia,** tending to show that the reflexes, especially of the triceps, in the upper extremities are modified as frequently and as early as the patellar reflex. He also gives emphasis to the fact that the hypesthetic thoracic girdle, first described by Lehr, is commonly found early, being one of the most constant sensory disturbances; also hypesthesia in the ulnar border of the hand, especially affecting the little finger and the ring-finger. He draws the conclusion that in the early stages of tabes the conception that the lesion is largely confined, in the majority of instances, to the lumbar cord must be revised, his tabulated cases giving proof that the cervical enlargement of the cord is commonly affected early. At the same time, in the cases which show early loss of triceps and patellar reflexes he commonly observes that myoidism is readily induced by the stroke of the percussion hammer across the long dimension of the muscle. [Investigation of the elbow reflexes in a large number of cases does not indicate that they are so commonly lost in early stages.]

Sabrazes and Fouquet [3] report an interesting case of tabes occurring in a woman, with early **lancinating trifacial pains,** for which she sought assistance from a dentist, who, on attempting to extract a tooth, produced a fracture of the alveolar process of both superior maxillary bones, breaking into both antrums and removing 11 sound teeth firmly implanted in the dental border. Motor and sensory disturbances in the limbs did not appear until 2 years later. The deformity might be mistaken for the perforating ulcer that occasionally occurs in the mouth in tabetics. The nature and possibility of the accident should be known to surgeons and dentists. [Trifacial pain is not absolutely rare, and is being recorded with increasing frequency now that the upper level types of tabes are more carefully studied.]

C. A. Oliver [4] contributes a clinical study on the **ocular symptoms** found in posterior spinal sclerosis. He emphasizes the important fact that for long periods of time, in spite of coarse objective changes in the nerve-head, even normal central vision may be present, but the color-fields are irregularly indented, and often notably diminished very early.

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[1] Rev. de méd., July 10, 1900.

[2] Deut. Zeit. f. Nervenh., July, 1900.

[3] Nouv. Icon. de la Salpêt., 1900.

[4] Am. Jour. Med. Sci., July, 1900.

J. C. Roux [1] in a recent work makes a study of the **sympathetic nervous system in tabes.** He has studied the large splanchnic nerves and the cervical and thoracic portions of the sympathetic chain in 7 cases of tabes. His examination has been confined to the medullated fibers, of which the small fibers are more numerous in the sympathetic system than the large ones, and these smaller fibers the author has found degenerated. He has determined that about one-half of the small medullated fibers disappear in tabes, while the larger fibers remain unaltered. This degeneration is the result of changes in the posterior spinal roots, whence the small fibers are derived, and from this region the small fibers in the cases examined had almost entirely disappeared. These fibers in the posterior spinal roots are supposed to have a sensory function, and their alteration may be associated with visceral crises and other symptoms so commonly encountered in tabes. The author also finds that in tabes a blow in the epigastric region may give rise to no discomfort, and believes that this loss of sensation is due to degenerative changes in the solar plexus and splanchnic nerves.

L. von Dydynski [2] details a case of **tabes in a child,** and refers to the literature. His conclusion is that the etiology of tabes in children, the pathogenesis, is absolutely dependent upon a syphilitic element, without which it can not exist.

A. Elsching [3] has made a careful microscopic and pathologic examination of the **changes in the fundus** in 2 cases, 1 of tabes and 1 of disseminated sclerosis. In **insular sclerosis** he states that the atrophy is the result of the interstitial infiltration occurring in spots and small patches in the optic nerve, resulting in the increased formation of connective tissue and the destruction of nerve sheaths and nerve-fibers. This process he considers identical with that occurring in the central nervous organs in the same disease. In **tabes,** however, he found a complete primary atrophy of the ganglion cell, also of the retina and of the nerve-fibers, and a diminution in volume of the entire optic nerve, in cross-section, with increase of the neurogliar tissue in the optic papilla, while in the remaining parts of the optic nerve he found a partial disappearance of the nerve-fibers; thickening and sclerosis of the blood-vessel cells were also present. The most marked changes were in the papilla, and diminished in intensity toward the region of the optic tracts. It would, therefore, appear that optic atrophy in tabes resembles the degeneration of the other sensory neurons arising from the spinal ganglia.

C. J. Aldrich [4] reports an interesting case of tabes in a woman presenting **symptoms referable to the sacral cord.** There was a cannon-

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[1] Lesions of the Sympathetic Nervous System in Tabes, and their Relation to the Disturbance of Visceral Sensibility, Paris, 1900.

[2] Neurol. Centralbl., April, 1900.

[3] Wien. klin. Woch., 1899.

[4] Med. News, Nov. 25, 1899.

ball sensation in the rectum, with loss of sphincter control, and fulminating pains in the perineum and over the area of tactile anesthesia. The sensory disturbance occupied a saddle-shaped area involving buttocks, perineum, and stripes down the back of the lower extremities—the exact cutaneous distribution of the sacral segments. There were also gastric and laryngeal crises and a very rare clitoral attack, in which the patient confessed to having spells of intense sexual excitement, which caused her great distress. Crises of the clitoris are found by the author in only one other reported case—namely, by Pitres. The left eye was blind, but did not present atrophy a year after the blindness had been discovered.

**Syringomyelia.**—F. W. Langdon [1] describes a case of syringomyelia in a girl of 19 in which the atrophy was the most pronounced feature, rather closely simulating the deformity of progressive muscular atrophy of the scapulohumeral type. The case is unusual in the symmetric distribution of the atrophy and the youth and sex of the individual.

Nebelthau [2] reports an observation showing that **syphilis may be the cause of spinal gliosis** and syringomyelia. He makes reference to several similar cases recently published by different authors.

**Hematomyelia.**—Fischer [3] gives the history of a smith 32 years old, a heavy drinker, but without luetic or other illness, who suddenly lost power in his right arm when using the hammer. This increased, and the lower right extremity was finally involved, feeling heavy and being more or less uncontrollable. He died 4 days later, the brain and cranial nerves being uninvolved to the last. Death occurred from involvement of the respiratory apparatus. In the lower portion of the cervical cord a hemorrhage 7 cm. long, most marked in the posterior columns, was found. A number of small hemorrhagic points were also discovered in the brain and cortex, and one in the neighborhood of the fourth ventricle. There was some hyaline degeneration of the middle coat of the blood-vessels, but no atheroma. The hemorrhage is supposed by the author to be primary, and induced, in all probability, by the extreme muscular labor of the patient and his drinking habits.

Pearce Bailey [4] attaches much importance to the probability of hemorrhages of greater or smaller size occurring in the substance of the cord after spinal injuries. He recalls the cases of Thorburn, in which 6 were examples of primary spinal cord hemorrhage out of 21 cases of traumatism of the cord; 1 out of 7 reported by Parkin; 2 out of 22 reported by Stolper. Inasmuch as many of these hemorrhages are minute and the tendency to recover is pronounced, the probability of their frequency is enhanced. The situation of the hemorrhage is usually in the gray matter, but the white matter does not entirely escape. When hemorrhage takes place in the white matter, it is usually in the dorsal column, behind the gray commissures. The size of the hemorrhage varies; it may extend for several inches, completely hollowing out the cord; in other instances it may be punctate. The cervical and lumbar swelling and the conus are the favorite sites, but in rare instances a punctate, widely disseminated hemorrhage may take place throughout the entire length of the cord, as shown in a case reported by the author. He considers that hemorrhages within the cord are beyond the reach of surgical aid, and as there is a relatively favorable prognosis when the diagnosis can be made, operation should not be considered. Rest in bed is usually made obligatory by the paraplegia; but if it is not compelled by the condition, it should nevertheless be insisted upon. Thereafter massage, electricity, passive movements, resistive movements, and similar manipulations are to be persistently and seriously undertaken.

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[1] Report of clinical lecture delivered at the Cincinnati Hospital, Dec. 22, 1899.

[2] Zeit. f. Nervenh., Feb., 1900.

[3] Festschrift, Fiftieth Anniversary of Dresden Hosp., 1899.

[4] N. Y. Med. Rec., April 7, 1900.

J. H. Lloyd [1] reports a well-observed case of hematomyelia. The motor symptoms suggested a lesion as high as the third cervical segment. The phrenic nerve was evidently involved, but as it is represented in the second, third, and fourth cervical segments, it was only partly disabled. Death in the case resulted from respiratory difficulties, partly due to the failure of the diaphragm, perhaps by progressive involvement of the phrenic cells in the cervical cord. The sensory symptoms also indicated a lesion as high as the third cervical segment, and this location was demonstrated postmortem. The knee-jerks were not abolished, but exaggerated, and the lesion was found to be partial as far as the transverse cord section was concerned, a belief expressed at the bedside. There was hyperhidrosis above and anhidrosis below the sensory boundary-line.

W. F. Becker [2] reports 3 cases of hemorrhage of the cord. In the second case the diagnosis was verified by postmortem examination. In all three the onset was sudden, following protracted effort or a concussion, as in the second case.

**Myelitis.**—Schiff [3] reports a case of **hemorrhagic myelitis** occurring in typhoid fever. The symptoms were those of acute ascending paralysis, incontinence of bladder and rectum preceding other symptoms by several hours. The patient died within 24 hours, during which time a bed-sore the size of the palm developed over the sacrum. Autopsy showed the spinal cord at the level of the fourth, fifth, and sixth cervical nerves to be dark, swollen, and soft, and the normal structures obliterated. Small, irregularly disseminated recent hemorrhages throughout the spinal cord and hemorrhagic infarction of the lower part of the spinal segments with neighboring vascular dilation were demonstrated.

Charles G. Stockton [4] reports a case of **acute ascending paralysis** in which there was **hematoporphyrinuria,** confirmed by J. B. Ogden, of Harvard Medical School. The author queries whether there is a direct relation between acute inflammatory processes of the cord and this condition of the urine. It has been noted by Ogden in a case of post-syphilitic neuritis, but in this case, as well as in one reported by Stockton, trional had been used, though under such conditions that it was impossible to see any relation between the drug and the renal symptoms.

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[1] Jour. Nerv. and Ment. Dis., Feb., 1900.

[2] Med. Rec., Aug. 18, 1900.

[3] Deut. Arch. f. klin. Med., 1900.

[4] Am. Jour. Med. Sci., July, 1900.

**Tumor of the Spinal Cord.**—Boettiger [1] reports the case of a woman of 65 who presented symptoms of disease of the spinal cord giving rise to a diagnosis of tumor. Operation was done by Krause and the neural canal was opened for a distance of 6 cm. in the region of the seventh and ninth dorsal vertebræ, where a tumor the size of a hazelnut was uncovered. It was a psammoma, and had decidedly compressed the spinal cord at the level of the eighth dorsal segment. A good recovery from the operation was obtained and the patient gradually improved. Control of the sphincters and natural sensations in the lower extremities promptly followed, and nerve-root symptoms subsided.

**Progressive Muscular Atrophy.**—Abadie and Denoyes [2] report an instance of progressive muscular wasting, apparently belonging to the dystrophic variety, and showing, in addition, qualitative changes in the electric responses obtained upon careful examination, enabling them to state positively the presence of reaction of degeneration. The contour of the muscles and general appearance of the patient conformed absolutely to the picture of pseudohypertrophic paralysis, but there was the absence of family history, though some of the stigmata of degeneracy were present in the patient, such as the Gothic palate, irregularly implanted teeth in abnormal number, deformed ears, chronic blepharitis, thoracic malformation, inguinal hernia, cryptorchidism. [The case is of extreme importance as presenting a transitional type between the spinal and so-called myopathic varieties of muscular wasting.]

F. Pick [3] furnishes a very interesting, instructive, and critical article upon this subject, based upon a full clinical and anatomic examination of one case, and clinical study of a number of other cases, with microscopic investigation of portions of muscles removed during life. In the opening pages he brings out strongly the fact, shown by well-authenticated instances, that there are all variations between the numerous varieties of muscular atrophy, and that clinically it is absolutely impossible in some instances to decide whether the spinal cord is affected or not; whether the case is one of so-called muscular dystrophy or belongs to the spinal variety. His patient was a man of 52 years who had always been well up to 2 years before he entered the hospital. At that time he presented well-marked atrophy of some of the muscles of the neck (especially the sternomastoids), of the small muscles of the hand, of the deltoids, and of the thenar eminences. There were also fibrillar contractions in the tongue, and later the speech was disturbed. A clinical diagnosis of spinal muscular atrophy was made. The anatomic examination showed the spinal cord, the brain, and the peripheral nerves to be absolutely intact, with the exception of a degenerated bundle in the spinal accessory, associated with the sternomastoid, which for a long period of time had been absolutely useless from complete atrophy and degeneration. The affected muscles showed atrophy and lipomatosis, and the muscle spindles were normal. The case, therefore, and the technic, as given, seems to have been perfect in all details, corresponds to the descriptions of primary myopathic atrophy. From the conditions of the muscle spindle and a study of the literature of the subject he supports the idea that they are purely sensory in function, and have directly to do with the muscle sense. In pieces of muscle taken from well-marked cases of family myopathy, and from a case of pseudohypertrophic paralysis occurring at the age of 46, but which had presented some weakness of the legs from birth, he found the condition of the vessels, nerves, muscle-fibers, and muscle spindles to be identical with those presented in his first case. He calls attention to the contributions of Leonowa and Carl and Gustav Petrén, who report a number of instances in human fetal monsters in which there was absence of the spinal cord, and yet the muscular apparatus was well developed, as tending to show the independence of muscular development in opposition to the theory of trophic dominance of the spinal cord. He also refers to the fact that new-born animals present a very much larger number of muscle-fibers than adults, and to the so-called "sarcolysis" that occurs after birth. Meek, for instance, found in kittens examined at the ninth, twentieth, and two hundred and fortieth day of life that the biceps contained respectively 83, 514, 64, 100, and 37,830 muscle-fibers, while the biceps of a 3-year-old cat presented but 22,858. He raises the question whether this physiologic atrophy or diminution in the number of muscle-fibers may not in some instances, from hereditary or prenatal causes, become progressive dystrophy, and finally concludes that the primary myopathic nature of these dystrophies must still be maintained. A very interesting portion of his report concerns the spinal accessory nerve previously mentioned. The bundle supplying the sternomastoid showed degeneration, which he believes to have been secondary to the complete atrophy of the muscle. He thinks that in other instances of progressive muscular atrophy the modifications of the anterior horn cells are probably secondary to the dystrophy, and of a nature somewhat similar to the changes which take place after amputations, being, however, usually less in degree, owing to the fact that the parts retain more or less of their muscle-fibers and muscle sense and all the centripetal sensory influences.

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[1] Neurol. Centralbl., July 15, 1900.

[2] Nouv. Icon. de la Salpêt., Aug., 1900.

[3] Deut. Zeit. f. Nervenh., Bd. XVII, 1900.

## NEUROSES DEPENDENT UPON INFECTION.

**Tetanus.**—C. F. Cuthbert[1] reports a case of tetanus, appearing 11 days subsequent to a contused wound over the shin, in which intracerebral injection of antitetanus serum into both frontal lobes, supplemented by a number of subcutaneous injections, while apparently controlling the disease temporarily, preceded a fatal termination by delirium and presumably heart failure, after a struggle.

Eichorn,[2] of Munich, describes a case of tetanus of moderate severity originally published by F. Schultze, of Bonn. An injection of antitoxin serum was made by lumbar puncture, and was followed by an extension of the tonic condition to muscles not previously involved. Recovery subsequently took place, and the authors believe that the lumbar injection is preferable to the intracerebral process. They assert that relatively larger amounts may be used when it has been found by subcutaneous employment that the serum does not cause fever or inflammation.

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[1] Brit. Med. Jour., Nov. 18, 1899.

[2] Centralbl. f. klin. Med., Sept., 1900.

Loeper and Oppenheim [1] report 5 cases of tetanus. Four were treated with antitetanus serum and recovered, while a fifth was treated with the trephine and intracerebral injections of serum and died. They strongly favor the hypodermic method, but express the idea that intravenous injection may prove even more desirable.

**Chorea.**—Westphal, Wassermann, and Malkoff [2] report in great detail a case of articular rheumatism followed by chorea, complicated by endocarditis and nephritis, in which they succeeded in isolating from the blood, the brain, and the endocardial vegetations a streptococcus capable of inducing arthritis in lower animals. The pyogenic nature of articular rheumatism and chorea has also received support from other observers. Mircoli [3] relates that pyogenic cocci were found in the joints of 14 out of 17 cases of rheumatic chorea, staphylococci preponderating 11 times, and Diplococcus lanceolatus 3 times. [The pregnant inference from these investigations is that acute rheumatism is to be looked upon as a variety of pyemia without suppuration, and chorea as a manifestation of the cerebral localization of the pyemic process.]

Gilles de la Tourette [4] reports 2 cases of supposed **chorea in pregnant women.** The first was aged 18, was 8 months pregnant, and had presented involuntary movements in the left side of the body for a month and a half, these movements following an exposure to cold and supervening upon an almost delirious state. At the time of observation the patient was melancholy, was addicted to coprolalia, and her left side was hyperesthetic. The mental condition, it was found, was of several years' duration. The patient had been a sleep-walker at the age of 8, and had facial tics since she was 12 years old. He believes that hysteria and the tic disease explain the involuntary movements of her left side. In a second observation a young woman in the first months of pregnancy presented incoordinate movements of the left side. An attentive study of her antecedents and an examination showed that these movements were attributable to the maladie des tics. He concludes that the chorea of pregnancy should be restudied; that it is probably made up of very different elements. Inasmuch as the chorea of Sydenham does not exist after puberty, and chronic chorea is extremely rare, hysteria and the maladie des tics will probably be found to cover the majority of the incoordinate movements presented by pregnant women.

Krafft-Ebing, [5] basing his study upon 200 cases of the disease, does not believe that the neuropathic soil is an essential to the development of chorea, since in 75 of his cases a nervous predisposition was present in only 50. In cases in which the chorea developed after pyschic trauma a neuropathic heredity was much more manifest. In 66 such cases a hereditary tare existed in 64. Cases referable to such mental trauma presented symptoms within a very few days after the reception of the mental shock. In 59 of the 200 cases the exciting cause could not be ascertained. The author believes that side by side with cases of chorea occasioned by infection there are others in which the disease is of nervous origin, developing under the influence of mental shock.

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[1] Arch. gén de méd., April, 1900.

[2] Berl. klin. Woch., 1899.

[3] Berl. klin. Woch., 1900.

[4] La Semaine méd., 1899.

[5] Wien. klin. Woch., 1899.

**Tetany.**—E. H. Burkhardt[1] reports an observation in which **retention of the urine** was the predominating symptom. The patient, a weak infant of nearly 3 years, living under bad hygienic conditions, the child of an alcoholic, presented cachexia and rickets. There had been convulsions and digestive disturbance during the previous months; the bladder was full and contained albumin. The day after admission the bladder reached the umbilicus, and convulsions occurred. On the following day retention was again marked, requiring catheterization, when for the first time carpopedal spasms were noted. Passing contractures in the distribution of the facial nerves were observed, with wrinkling of the brow and occasional difficulty in swallowing. Chvostek's and Trousseau's signs were easily demonstrated. The knee-jerks were abolished. The urinary retention was undoubtedly dependent upon contracture of the vesical sphincter, and presented a distinct obstacle to the catheter. Somewhat similar instances have been reported by others, and emphasize the importance of examining the condition of the bladder in all cases of tetany.

G. Kirchgasser[2] contributes a very important article on the **relation of tetany to rickets** and laryngismus stridulus. Grouping the statistics of a number of other reporters—Boral, Fischl, Cassel, Bendix—with his own, he analyzes the enormous number of 49,425 cases, of which number 19.03 % presented rickets, 2.08 % tetany, and 4.41 % laryngismus stridulus. In 283 cases of tetany he found rickets to be present 226 times—a ratio of 79.81 %. In 443 cases of laryngismus stridulus rickets was present 398 times—89.04 %. Turning his attention to the seasonal conditions, he finds that rickets reaches its highest curve in the fourth and fifth months of the year, tetany in the second and third months, and laryngeal spasm in the fourth month. From statistical studies he concludes: that in Germany rickets is present in two-fifths of all children treated in polyclinics; that of those presenting tetany and laryngospasm four-fifths, or double the proportion of patients treated, present rickets; that laryngospasm is twice as frequent as tetany, and that it has a closer relation to rickets than has tetany; that laryngospasm and tetany are combined in about half the cases of tetany and in a fourth of the cases of laryngospasm. The author also reports 2 cases of tetany dying with laryngospasm in which careful examination of the brain and spinal cord were made by the most careful methods. By Marchi's method more or less well-marked changes were observed in the anterior and posterior spinal and cranial nerve-roots. According to Weigert's method, nothing abnormal was found, and sections of the radial and ulnar nerves were negative.

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[1] Jahrb. f. Kinderh., 1899.

[2] Deut. Zeit. f. Nervenh., 1900.

## PSYCHONEUROSES.

**Migraine.**—Krafft-Ebing [1] reports a number of cases of migraine with **transitory mental disorder** occurring as a part or as an alternative of the migrainous attack. In the most demonstrative instance the patient presented an ocular form of migraine with scotoma and visual hallucinations. In certain attacks when the scotoma disappeared the violent headache continued, and the "curious stage" appeared, lasting about 5 minutes. During this stage she feels as if out of her mind, does not know herself nor her husband, has a vague feeling of impending insanity, is greatly confused, does not utter a word, apparently is unable to do so, has the feeling that she is persecuted by some one. She does not lose consciousness, but the feeling is extremely painful, with the fear that she is losing her reason. She has had similar attacks at intervals for 2 years. In other instances quoted by the author he considers the mental disturbance to be a psychic epileptic equivalent.

R. Paderstein [2] makes a contribution to the subject of **ophthalmoplegic migraine,** and presents a number of cases. He concludes that there is an idiopathic form of illness in which migraine is combined with attacks of palsy of the eye muscles, and that the term ophthalmoplegic migraine is most desirable, as the old designation of oculomotorius paralysis is misleading. Whether the ophthalmoplegic migraine is a form of ordinary migraine or a disorder by itself is not yet determined in the absence of definite knowledge of the pathology of both disorders.

**Facial Tic.**—E. W. Stevens,[3] considering the relation of facial spasm to errors of refraction, concludes that in all cases the refraction and muscle balance should be carefully examined; that refraction should be determined in complete mydriasis and full correction ordered; and that it is not sufficient to get rid of the cause, for the nerve-centers, having acquired a vicious habit, do not recover normal conditions until systematic treatment has been pursued. [In this third consideration—namely, the acquirement of a vicious habit—the author hints at, but does not apparently recognize, the all-important factor of mental disorder, though his systematic treatment no doubt serves a good purpose by the mental impression produced.]

**Epilepsy.**—Richet and Toulouse announced before the Paris Academy of Sciences in November, 1899, that they had treated 30 female epileptics by the **deprivation of salt** from their food, thereby, as they supposed, rendering the nervous tissue more susceptible to the absorption of medicinal salts. Thus, 30 grains of bromid of sodium a day, administered under the conditions referred to, prevented the epileptic attacks in severe cases, and some patients had shown no recurrence for 6 months. They suggest calling this régime the metrophic method, and think that it may possibly apply also to affections requiring quinin, digitalis, etc., as well as to those in which the alkaline salts are administered. The saltless food had no bad effect on the patient.

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[1] Alienist and Neurol., Jan., 1900.

[2] Deut. Zeit. f. Nervenh., 1899.

[3] Am. Jour. Med. Sci., Jan., 1900.

P. Naecke [1] describes the method of treating epilepsy recently advocated by Toulouse and Richet, which consists in the withdrawal of all salt from the food, the supposition being that the tissues deprived of sodium chlorid the more readily take up the bromids which are employed, owing to the lack of other halogen bases in the dietary. No definite statements as to results are given, but the impression of this writer seems to be that the measure is one of value.

The **toxic theory** of epilepsy, which has received so much attention, is investigated, so far as the condition of the urine is concerned, most thoroughly by J. J. Putnam and Franz Pfaff.[2] Twenty-nine specimens of 24 hours' urine, coming from 2 patients, were examined. The first patient had 5 seizures during the period corresponding to the analysis; the second patient had 2 seizures. The result did not bear out the contention, as regards either the low excretion of uric acid before the attacks or the high excretion after them.

Charles Hill [3] has made a trial of **suprarenal extract** in the treatment of this neurosis. Hill found that it increased the elimination of urine, and was thereby led to make trial of the drug. Glycerin extract of the fresh gland was made and was found to be superior in constancy of its effects to other preparations. One dram of the solution represented 1 grain of the gland. After a year's experience he says, "I would consider it a success compared with any and all other plans of treatment if there was nothing more in its favor than the striking improvement in the mental and physical condition of the patients," which was generally and favorably influenced.

Seglas and Heitz [4] report upon the treatment of epilepsy by the **opium method of Fleichsig.** Twenty-two patients were subjected to the treatment at the Bicêtre Hospital. The opium was given, according to the well-known rule laid down by Fleichsig, in increasing doses until about 15 grains were administered in the 24 hours, and then abruptly withdrawn, bromid being substituted. The authors conclude that this treatment is not tolerated by many patients, and that its administration necessitates a care and superintendence that can be furnished only in fully equipped hospitals, and even then is always difficult and sometimes very dangerous. Its contraindications are far from being compensated by the benefits, which are always inferior to those of simple bromid administration. Among the disadvantages are vomiting, diarrhea, albuminuria, oliguria, shallowness and slowness of respiration, rapid loss of flesh, myosis, somnolence, hebetude, delirium, and other psychic disorders. They also refer to 8 deaths in 200 cases treated by Bratz.

**Neurasthenia.**—S. Lubetski [5] contends that the experiments of Mosso and d'Anjel prove that the weakening of the arterial tone in neurasthenics produces a **cerebral vasodilation**; that venous stasis augments the volume of the brain, which within the rigid confines of the cranium produces slight compression, whence arises the headache. He also maintains that clinically all measures which decongest the brain either dispel the headache or reduce its intensity, while opposite means add to it. Further, he says that cranial thermometry proves directly that the brain in neurasthenia is in a congested state, the temperature being somewhat more elevated than in healthy individuals, and the maximum of the headache corresponds to the point where the temperature is the highest. Moreover, the temperature diminishes with the recession of the headache. He has found an addition of 2 degrees in the local temperature between conditions in which headache was present or not.

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[1] Neurol. Centralbl., July 15, 1900.

[2] Am. Jour. Med. Sci., Aug., 1900.

[3] Bull. Mount Hope Retreat, 1899.

[4] Arch. de neurol., Aug., 1900.

[5] Thèse de Paris, 1899.

Romeiser and Collins [1] contribute an article on the **condition of the blood** in neurasthenia—a subject that has received considerable attention abroad. They do not find, however, that there is anything peculiar in the blood of neurasthenics, but state that in general the condition of the blood corresponds to what might be expected in such a bodily condition as attends neurasthenia. Great variation was often observed in individual cases, and no two cases were exactly alike, as regards symptomatology, severity of the disease, or blood state. They found that the blood changes corresponded rather to the objective than to the subjective clinical picture.

Karl Petrén [2] inquires as to the social state of neurasthenia and confirms the statement that the disease is as common among hand workers and laborers as it is among those more fortunately situated. It also appears to be slightly more common among men than among women.

Andre [3] calls prominent attention to the **dyspnea of neurasthenics.** He refers particularly to the feeling the patients express of being unable to get sufficient air into the lungs, often making statements that the air "does not seem to get down into the lungs or reach the right spot." He also notes the significant fact that if their attention be strongly diverted, or they are in any way distracted from the subject in hand, the difficulty of breathing subsides. Moreover, it is not attended by any objective inconvenience or respiratory difficulty; cyanosis, rapid breathing, and other similar defects do not accompany it; neither is the pulse accelerated unless the patient's anxiety reaches a point where the cardiac action is modified by the emotional state.

**Hysteria.**—F. Walter [4] claims to have found a valuable remedy for hysteria by the **exhibition of derivatives of the colon bacillus** taken from a noninfectious source. He limits the value of the treatment, however, to the active convulsive type, and claims that the symptoms disappear in from 24 to 48 hours, the patient being restored to health, so far as the hysteria is concerned, but the same result does not follow in what he calls latent hysteric phenomena. He would class this remedy as a specific. The culture is prepared upon the surface of agar-agar in Petri dishes, and when well developed may be scraped off, suspended in water, or mixed with some indifferent substance and administered in capsules. The products of one dish make from 3 to 6 doses. [This treatment has found no supporters so far as known.]

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[1] Medicine, Nov., 1900.

[2] Deut. Zeit. f. Nervenh., Aug., 1900.

[3] Rev. Neurol., Aug., 1900.

[4] N. Y. Med. Jour., July 21, 1900.

Krafft-Ebing [1] presents a very instructive case of **hysteria mimicking paralysis agitans** lasting for years, with the characteristic tremor and the highly distinctive handwriting, a sample of which is published with the report. After careful study and examination it was finally classified as a hysteric condition secondary to mental shock, with a tremor identical to that presented by paralysis agitans. The disease had lasted 23 years without any particular modification. The principal hysteric stigmata in the case were slight hyperesthesia to cold, associated with left-sided hemihyperalgesia, and the mental shock was furnished by the sudden death of an only daughter.

J. Abadie [2] presents an interesting study of **polyuria and frequent urination** in hysterics, and reaches the conclusion that they are susceptible of cure by both direct and indirect suggestion.

G. Comar,[3] struck by the fact that hysterics often present sensory stigmata in the region of the organs that are disturbed, investigated the **sensibility of the head** in the neighborhood of the cortical centers related to body segments and limbs, affected by the neurosis, and believes that in many such instances hyperesthesia or anesthesia and headaches of corresponding location can be detected.

P. Sollier [4] expresses the belief, and presents cases to substantiate the thesis, that areas of the cortex and of the corresponding scalp and skull show disturbance in the way of hyperesthesia, subjective pains, and general or localized headaches related to the body and limbs affected by hysteric disturbances. The internal organs also present their spots of tenderness on the head. The stomach he considers to be represented on both sides of the skull, but more particularly on the left side, about 5 cm. behind the biauricular line—that is to say, over the superior parietal lobule. For the heart he has only observed a median point a little in front of that for the stomach, while the centers for the bladder and intestine are situated near the median line and 5 cm. or 6 cm. behind that for the stomach. The center for the larynx corresponds nearly to the foot of the third frontal convolution, and that of the respiratory apparatus above it is in the neighborhood of the second frontal, while those for the genitals seem to be situated at the foot of the first frontal.

Brissaud [5] sustains the thesis that **polyuria** is a hysteric manifestation. In other words, that simple polyuria is not related to diabetes, but is always significant of the hysteric state.

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[1] Deut. Zeit. f. Nervenh., 1900.

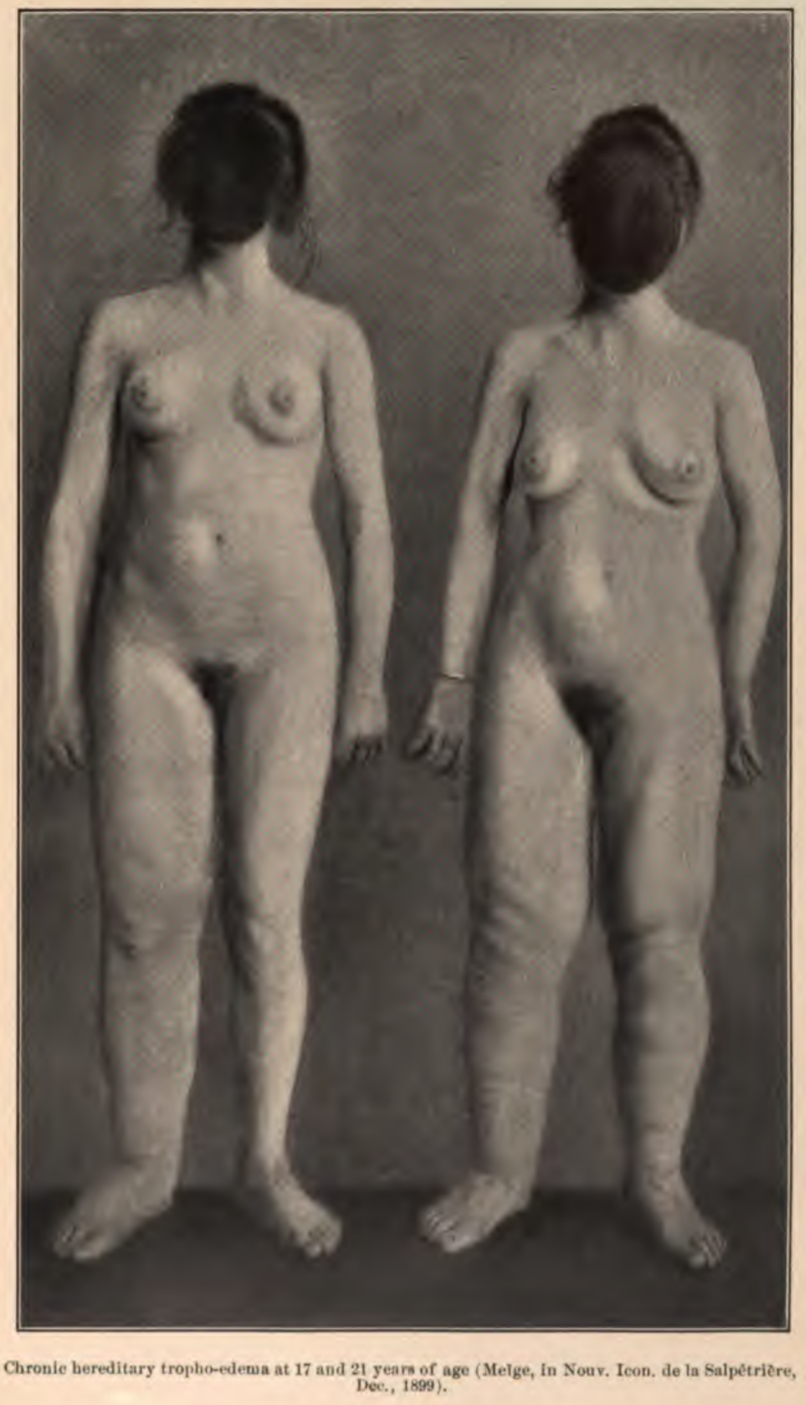
[2] Arch. de neurol., Mar. 19, 1900.

[3] Rev. Neurol., June, 1900.

[4] Rev. Neurol., Feb. 15, 1900.

[5] Leçons sur la Maladie Nerveuse, 1899.

PLATE 5.



Chronic hereditary tropho-edema at 17 and 21 years of age (Meige, in Nouv. Icon. de la Salpêtrière, Dec., 1899).

## TROPHONEUROSES.

**Chronic Hereditary Tropho-edema.**—Henry Meige [1] describes a family in which edema affected 8 members, both men and women, distributed through 4 generations. Four of these cases were observed, and presented the same singular affection: namely, a chronic white, firm, and painless edema, appearing at the age of puberty and affecting especially the feet and legs and sometimes the entire lower members, generally on both sides. He also refers to a remarkable family reported by Milroy in 1893 in the "New York Medical Record," in which in 6 generations there were 22 cases.

**Acromegaly.**—Sainton and State [2] attempt to differentiate a form of acromegaly which is particularly marked by pains. From a study of 140 reported observations, pains are mentioned 70 times, sometimes situated in the extremities, sometimes in the spine, sometimes in the viscera; but pains in the members are the most common, and are generally symmetric, usually involving all 4 extremities. They would divide pains into osteo-articular, neuralgic, muscular, and tabetic, and pains of the extremities. [While it does not seem necessary to make a distinction, as the authors would do, the importance of pain in acromegaly is emphasized by their contribution, and becomes a symptom of some diagnostic value.] They report a case in which they note certain changes in the spinal cord, which they epitomize as follows: (1) The presence of osseous infiltration of the dura mater, with the production of calcareous seeds on its internal surface; (2) cord lesion of the nature of degeneration, principally in the columns of Goll. They would conclude that these osseous lesions and changes in the cord are associated with the pains that mark so many cases of acromegaly.

Woods Hutchinson [3] contributes an able article to the study of **acromegaly and giantism** and the function of the pituitary gland. He concludes: (1) That the pituitary body is still functional. (2) That disturbances of its metabolism are the principal factors in both acromegaly and giantism, the difference between results being simply due to the stage of individual development at which the disturbance of the function begins. (3) That the nature of the overgrowth in both these diseases is primarily on the order of a pure functional hypertrophy; later, however, losing something of the definiteness of its impulse, and either producing immature tissue of a mixed type, or resulting in hemorrhagic exudation, with either cyst formation or complete breaking down of the tissue mass. (4) That it seems probable, although upon this head the evidence is still uncertain, that some part is played by this body in "dwarfism," rickets, and the dwarf forms of cretinism. (5) That a reflex disturbance of its function may possibly underlie the dystrophy accompanying pharyngeal adenoids. (6) That it would appear to be a sort of "growth center" or proportion regulator of the entire appendicular skeleton.

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[1] Nouv. Icon. de la Salpêt., Dec., 1899.

[2] Rev. Neurol., April 15, 1900.

[3] N. Y. Med. Jour., July, 1900.

William M. Leszynsky[1] states that **preparations of the pituitary gland** used empirically as a remedy in acromegaly have proved absolutely without value; that there are neither rational nor logical grounds for their administration in that disease. This conclusion he bases upon the fact that instances of tumor involving the pituitary prehypophysis, without symptoms of acromegaly, are on record; and, further, if it be accepted that the secretion of the glandular portion of the pituitary acts, as claimed by some, to stimulate directly the connective-tissue cells, and by persistent action to increase their growth, the use of the pituitary as a therapeutic agent would be directly contraindicated in a disease like acromegaly, where this increase has reached enormous proportions.

**Graves' Disease.**—C. M. Allan[2] suggests the treatment of Graves' disease by the **administration of bile** through the mouth, hypodermically, and deeply injected into the thyroidal mass. In the case of a married woman of 50 years who, after about ten years of nervous disturbance attributable to Graves' disease, developed a large thyroid after an attack of grip, the author gave, altogether, in the course of 5 weeks, 48,000 grains of bile, 3640 grains of which were injected hypodermically; and of this amount 1000 grains were injected into the substance of the thyroid. First the exophthalmos disappeared, then the rapid pulse, and then the tremors. None of the symptoms attributed to the circulation of bile in the blood occurred as a result of this treatment. The author does not venture to say whether the reduction of the pulse is analogous to that produced by cholemia. He feels certain, however, that a feeling of well-being and improvement in the nervous, circulatory, and digestive systems was induced by the treatment. Six months later the patient had retained all the improvement and was better, though the thyroid gland was still somewhat enlarged. A second case, a woman 45 years of age, was said to be suffering with change of life, complained of nervousness, palpitation, debility, diarrhea, and insomnia, and looked frightened. No exophthalmos was present; there was slight enlargement of the thyroid, noticeable only on examination. The pulse was from 120 to 140 and there were tremors in the upper limbs and weakness in the legs. A large amount of urine was passed, but no albumin. Diarrhea was paroxysmal. Bile (240 grains) was administered every 4 hours by the mouth, and appeared in the urine and dejecta in 3 days, and was reduced to 8 grains thrice daily. In the second week injections were given. In all, injections being given daily for 10 days, 3000 grains were employed. Only one injection of 320 grains was made into the substance of the thyroid, owing to its small size. Four grains night and morning were administered for 2 months, at which time the tremor had disappeared and the heart had become steady. She had occasional attacks of diarrhea, especially if frightened. She was eating and sleeping well and had gained 6 pounds.

S. Popoff[3] reports a case in which a patient with well-marked evidence of the disease had a number of **hemorrhages** from the uterus, nose, gums, and lips. Especially was the hemorrhage free from the nose and uterus. A second case presented hemorrhage from the uterus and nose, and occasionally from the throat. The author is inclined to think that hemorrhages in some of the obscure forms of Graves' disease may be a helpful diagnostic indication, and thinks that they occur more frequently than would appear from the descriptions of the disease and the literature of the subject.

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[1] Med. Rec., June 30, 1900.

[2] Lancet, Aug. 26, 1900.

[3] Neurol. Centralbl., April, 1900.

Adami[1] deals at considerable length with the subject of goiter and its relation to Graves' disease. He believes that the essential cause of Graves' disease is different from that of ordinary goiter in that Graves' disease primarily depends upon some nervous or other stimulus acting on the thyroid gland and leading to increased activity. The sudden liberation of some thyroidal secretion serves to explain the paroxysmal disturbances in Graves' disease and also in ordinary goiter. Just what this primary nervous disturbance is or whence it arises is left for speculative imagination.

Chas. Ashard[2] presented several patients whose condition had been rendered worse by bilateral section of the cervical sympathetic. In one instance vitiligo had developed after the operation, but as this is sometimes noted in exophthalmic goiter, it is perhaps not attributable to the operation.

Dupre and Guillain[3] report a case in which Graves' disease, scleroderma, and tetany were all present.

## NEUROSES MARKED BY MOTOR SYMPTOMS MAINLY.

**Myotonia.**—Von Bechterew[4] contributes the suggestion that myotonia is due to a self-poisoning, to which conclusion he is more forcibly drawn by having observed a case in which myotonia was associated with gout.

**Myoclonia.**—Herman Lundborg[5] reports 5 cases, and believes that the primary cause of Parkinson's disease is an affection of the thyroid gland, and also that family myoclonia is caused by disease of the thyroid gland. In one family he found several cases of myoclonia and 5 cases of Parkinson's disease. He also reported a case of Parkinson's disease with myxedema.

**Paralysis Agitans.**—Krafft-Ebing[6] reports 7 cases of alleged traumatic origin—4 men and 3 women—out of a total of 110 cases, embracing 67 men and 43 women. In 5 of the 7 cases the trauma was a contusion or a sprain, in 1 there was contusion with freezing of the entire extremity, and in 1 contusion associated with a broken rib. The author emphasizes the alleged fact that if the disease is traumatic it always begins at the location of the trauma, while the nontraumatic variety begins in the distal portion of the extremity. The author urges the necessity of considering, in early cases, the possibility of ascending neuritis as the cause of traumatic paralysis agitans, though this was not found in any of his cases. In some of his cases Krafft-Ebing was able to exclude the mental factor of shock, on which Gowers lays so much stress. [Taken as a whole, however, the cases are not conclusive.]

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[1] Montreal Med. Jour., Jan., 1900.

[2] Rev. Neurol., Aug., 1900.

[3] Bull. de la Soc. méd. des Hôp., May, 1900.

[4] Neurol. Centralbl., Feb., 1900.

[5] Hygeia, 1900.

[6] Wien. klin. Woch., 1899.

H. C. Gordinier [1] discusses the pathology of shaking palsy, for which purpose he collects reports of 24 cases of Parkinson's disease examined by modern methods, and concludes that no one can longer include the disorder among diseases of the nervous system of a purely functional character without anatomic basis. He bases this on the following considerations: (1) In all cases examined there was a uniformity and constancy in the results of the anatomic findings. (2) These involved the blood-vessels, neuroglia, and nerve-cells, resulting in a proliferation of the nuclei and thickening of the walls of the blood-vessels, with a proliferation of the neuroglia about the blood-vessels, forming by confluence patches or areas of perivascular sclerosis; pigmentation with degeneration and consequent atrophy of nerve-cells and nerve-fibers, the cells resting in dilated cellular spaces. (3) The spinal cord was in all cases most affected, and in a few cases was the only part of the cerebrospinal axis possessing pathologic changes, these changes being most marked in the gray matter and in the lateral and posterior columns of the cervical and lumbar enlargements. (4) The lesions, although similar in character, decrease in intensity brainward, the brunt of the affection being confined to the spinal cord. (5) The changes, while resembling those found in senility, differ from them in intensity, in the presence of typical patches of perivascular sclerosis, in the absence usually of general arteriosclerosis, and in being associated with a group of characteristic symptoms, which often appear long before senility, and when once begun continue to invade, without cessation or intermission, little by little the entire body, producing a clinical picture as distinct and characteristic as that of any disease, organic in nature, with which we are acquainted. The primary seat of the pathologic changes is doubtless in the blood-vessels in the form of endarteritis and periarteritis, with consequent proliferation of the neuroglia in the immediate neighborhood and the production of patches or islands of perivascular sclerosis, which are characteristic of the disease. The changes are, therefore, those of a chronic inflammation involving chiefly the spinal cord, medulla, and pons, and to a less degree the motor cortex, the peripheral nerves, and vessels undergoing similar but less intense changes. The author considers the tremor to be nothing more or less than exaggerated muscle tonus, or an amplification of the fine invisible tremor that is constantly present in health.

**Family Periodic Paralysis.**—Leo M. Crafts [2] gives the full clinical history of an instance of this peculiar family disorder and makes full reference to the recent articles on the subject. Careful investigation was made for the purpose of isolating, if possible, some toxic element which acted in the production of these family periodic palsies.

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[1] Am. Jour. Med. Sci., Dec., 1899.

[2] Am. Jour. Med. Sci., June, 1900.

The feces were carefully examined by A. F. Irwin, Professor of Pathology in Hamline University. From the first bowel movement following an attack an extractive was obtained the exact nature of which is not yet determined, but its injection into rabbits and guinea-pigs produced a paralysis, gradually disappearing in 48 hours, and seems to furnish the first definite information as to the exact nature of this interesting and rare condition. The author calls attention to certain peculiarities in the physical development of several of the reported cases. His own patient showed massive muscular contours, and the same has been noted in other cases. Excised muscle-fibers have been found normal in several instances. Oppenheim reported waxy degeneration; Goldflam, hypertrophy of fibers and rarefaction of the fibrillæ and vacuole formation. In his own case the author found that a portion of the muscle taken from the calf presented a distinct increase in the fibrous tissue and a moderate hypertrophy with vacuolation of some of the fibers. There seems to be a relation between the dystrophies, the myotonias, and this particular state, and the author suggests that the poison found and demonstrated in his report may be found, or that similar toxic agents may be discovered, in both myotonia and primary myopathy.

## MENTAL DISEASES.

**Melancholia.**—Vallon and Wahl[1] call attention to the fact that **idiomuscular contraction,** or myoidism, is a very common condition in melancholia. They found it in 32 cases out of 40, and rather more frequently among men than among women. Among the 32 cases presenting local muscular contraction, 8 were affected with gastro-intestinal disorders and 1 had albuminuria. One was a case of simple melancholia, 8 of melancholia with stupidity, 16 of delirious melancholia, 3 of melancholia agitata, and 3 were in the melancholic phase of alternating insanity.

**Insanity in Lead-workers.**—Robert Jones[2] furnishes a very important article, based upon a large clinical observation, regarding the effects of lead in the production of insanity. He summarizes as follows: (1) That lead-poisoning is a contributory factor in the causation of insanity, and that in lead-workers there is a higher average number of general paralytics than in others of the population. (2) That there is a tendency in these cases to cardiac, renal, and arterial degeneration, with complications due to syncopal or epileptiform fits. (3) That most cases present marked signs of anemia and ill health, with unsteadiness of gait and general impairment of muscular strength, and very frequently a history of temporary failing vision. (4) That the mental symptoms may be grouped among one or other of the following varieties: (*a*) Those in the nature of toxemia with sensory disturbances, and which tend rapidly to get well; (*b*) those with hallucinations of sight and hearing more chronic in their nature, and which may be irrecoverable; the delusions in this class are almost invariably those of being poisoned or followed about, and are in the main persecutory; (c) those resembling general paralysis with tremors, increased knee-jerks, and incoordination, and accompanied with listlessness amounting to profound dementia, but which tend to get well. (5) That in most lead cases presenting mental symptoms the tendency is to recovery unless the patient dies early. The discussion of his paper before the Section of Psychology of the British Medical Association gave voice to the very natural criticism that many of his cases of paretic dementia attributed to lead did not indicate that syphilis had been excluded, though the consensus of opinion was that lead-poisoning could easily produce a symptom-complex indistinguishable from general paresis.

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[1] Arch. de neurol., 1900.

[2] Brit. Med. Jour., Sept. 22, 1900.

**Acute Delirious Mania.**—John Turner[1] presents this subject in a paper which was read before the Section of Psychology of the British Medical Society. The paper is illuminated by a careful study of the pathologic anatomy, with especial attention to the disorganization of the cortical cells. The author concludes: "That all forms of acute delirious mania are of toxic origin, but while some are obviously caused by the introduction of a poison from without,—namely, alcohol,—others are caused by the absorption of septic material, while a third class are due to autointoxication, probably as the result of perverted metabolism, which the disordered nervous system is very likely the cause of. The fact that in these acute cases, occurring as they do in the prime of life, we almost invariably meet with fatty degeneration of the liver—an organ one of whose functions is most probably to prevent the introduction into the system of poisonous intestinal products—is in corroboration of this idea. It may be asked, If acute delirious mania is of toxic origin, why is it that some nerve-cells suffer, while others perhaps in their near neighborhood escape, though all must be subject to the same poisonous influence? Possibly it may be that those particular cells which are chiefly concerned in the delirious condition, those, at any rate, which happen to be in a state of great activity, are those which will feel the chief effect of the poison. If, as some hold, an accumulation of pigment in the nerve-cell is an expression of past (and probably recent) activity, then the fact that those cells which are the most pigmented are usually those most affected will to a certain extent bear out this contention. But as regards the action of a possible poison on the nerve-cell and its functions, the subject is too speculative for any advantage to be derived in discussing it. The cells just described are found here and there in many other forms of insanity which did not during life betray any symptoms of delirium, and even in some undoubted cases of acute delirious mania only a minor proportion of the giant cells are remarkably affected; but in those cases with high delirium and a rapidly fatal termination we generally meet a condition in which all the giant cells are profoundly altered, and I believe that from an inspection of such a section a tolerably accurate diagnosis could be formed of the mental condition preceding death."

N. Macleod[2] reports 2 cases of **acute mania** managed by what he denominates the **bromid sleep**—a plan devised by himself in the management of the morphin habit, and now applied to acute disturbances of mental derangement. The first case was a married woman of 40, two years past the menopause, who suddenly became violently insane. The first day 2-dram doses of bromid of sodium were given in half a tumbler of water, 4 times, and a large quantity of milk. On the second day she slept until 7 in the morning, and the greater part of the forenoon the delusional state continued. Bromid was given in 2-dram doses 4 times, as on the previous day. The third day the patient slept all night; muttered when aroused. On the fourth day she could not be aroused. On the fifth day she could not be aroused and had to be fed with a spoon. The sixth and seventh days showed the same condition. There was gradual emergence from the sleep, so that on the eleventh day she was still sleeping and failed to put out her tongue when asked, or otherwise to respond. There was gradual improvement until on the twenty-first day, without further medication, she had a good night; and though delusions were apparent, they were slight and easily dispelled. A few days later she was considered well. The second case was a married woman of 30, nursing an 8-months' child. Acute delirious mania was the diagnosis, of 7 days' duration. Seven drams of sodium bromid were given in 3 doses on the first day, by catheter through the nose; in addition to which she had 30 grains of chloral and a dram of potassium bromid. The second day she slept the greater part of the night, still refused food, and was restless and talkative. Seven drams of bromid were given, as before. The third day she slept all night and talked quietly through the day. Restraint was no longer needed; she took a little milk and ate a little fruit. Seven and a half drams of bromid were given before 4 P.M. The fourth day she slept all night and could not be aroused to wakefulness. The fifth day she could not be aroused. There was cellulitis around the left carotid, submaxillary, and neighboring regions; temperature, 104° F. The sixth day she was still sleeping, and died at 8 P.M. The author thinks that death was obviously due to septic poisoning starting in the condition of the mouth, and believes that if the septic mischief had not occurred, recovery might have been expected. The author also makes a summary of the cases in which he has used the bromid sleep in the management of the opium habit, and which have been referred to in previous numbers of the YEAR-BOOK. [This treatment is decidedly dangerous, as pointed out by Church,[1] and has numbered at least 4 fatalities in less than a score of cases where it was employed for maniacal or morphinomaniacal conditions. Three ounces in as many days can not be exceeded with safety.]

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[1] Brit. Med. Jour., Sept. 22, 1900.

[2] Brit. Med. Jour., Jan. 20, 1900.

**Organotherapeutics in Insanity.**—C. C. Easterbrook[2] reported very wide observations, extending over 6 years, in which he administered to patients suffering from various forms of insanity extracts of thyroid gland, the parathyroid bodies, the thymus, the pituitary body, the brain, the choroid plexuses of the brain, the spleen, the suprarenal bodies, the testes, the ovaries, the uterus, and the mammæ. For instance,

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[1] Chicago Med. Recorder, Aug., 1900.

[2] Brit. Med. Jour., Sept. 22, 1900.

130 cases were submitted to the thyroid treatment, smaller numbers being treated by the other extracts. As conclusions he states that, apart from the general tonic effect upon cell metabolism, it is extremely doubtful whether each organ possesses a separate secretion, as held by Brown-Séquard, though some organs probably have such a secretion, as, for instance, the active principles of the suprarenals, which powerfully increase muscular contraction but diminish tissue oxidation; also the thyroid, through its active principle iodothyrin, the effect of which is to stimulate cell catabolism or tissue oxidation. The author was supported by the opinion of other members of the Section in Psychology of the British Medical Society, and the consensus of opinion seemed to be that organotherapy is of extremely limited value in the treatment of the insane. Occasionally in circular forms of the disease the depressed period can be avoided by the use of thyroids, and occasional cases of melancholia can be toned up by thyroids and other extracts. They are rich in nucleins, through which a timely stimulation of cell metabolism is induced.

**Thyroid Treatment.**—William Mahon and Warren L. Babcock [1] review the results obtained from the treatment of 1032 cases of insanity with thyroid extract, and reach the following conclusions: (1) The dose of the extract depends entirely on the individual case. In some cases 25 grains 3 times a day will be necessary to bring about a circulatory or temperature reaction, while in others the same results may be had with the use of 5 grains t. i. d. Each case must be a law unto itself. (2) It is essential that the patient should be placed in bed to obtain the best results, and he should be continued there during the entire treatment and for a week following its discontinuance. (3) The treatment should be continued for at least 30 days. (4) We should not be discouraged by failure in the first administration, but should resort to two, three, or more trials, if necessary. (5) The most gratifying results in thyroid treatment are to be obtained in cases of acute mania and melancholia with prolonged attacks, puerperal and climacteric insanities, stuporous states, and primary dementia, particularly when these forms of mental alienation do not respond to the usual methods of treatment. (6) A high temperature reaction is not essential, as it was found that the average maximum temperature in the recovered cases among men was 99.6° F. (7) Physical improvement is the outcome in most cases, whether mental improvement takes place or not. (8) The proportion of individuals who recover under thyroid treatment and then relapse is less than the proportion that relapse after recovery from other methods of treatment. In the series of cases reported only one patient who recovered has relapsed. [The bed treatment advocated may be the potent element in the treatment here indicated. It is particularly in the class of cases in which thyroids give the "gratifying results" that bed treatment is most indicated—a class of cases, moreover, that inherently do well.]

**The Bed Treatment of Insanity.**—In the Section of Psychiatry at the Thirteenth International Congress of Medicine, in Paris, this subject was discussed at considerable length. Jules Morel [1] summed up the situation as follows: (I) Every lunatic admitted into an institution should first be placed in the observation quarter for thorough examination, physical and mental. (II) Confinement to bed should be employed in the following cases: (1) All cases of acute or chronic psychosis presenting intercurrent conditions of excitement and depression; (2) all in which there is any disturbance of the general nutrition; (3) all cases of patients incapable of behaving in accordance with the rules of ordinary life—those that are mischievous or refuse their food or show suicidal tendencies, etc.; (4) all cases in which there exists a complicating somatic affection of any gravity. (III) In order that the object of the bed treatment may be satisfactorily attained, it is necessary (1) that the institution should have a competent medical staff proportionate to the requirements of the service; (2) that the various physicians attached to the institution should live in it and have each a real share in the observation and treatment of the patients; (3) that no means of restraint should be used except in very rare and exceptional cases; (4) that the medical staff should undertake the professional instruction of the attendants and eliminate from among them all who are not entirely satisfactory; (5) that the staff of attendants should be composed of intelligent persons of irreproachable morals, sufficiently paid and having the prospect of a retiring pension after a certain number of years of service; (6) that the buildings and their offices should satisfy all requirements as regards comfort both for the patients and the staff.

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[1] Am. Jour. Insan., Oct., 1899.

**Idiocy.**—In the Section of Psychiatry at the Thirteenth International Congress of Medicine, in Paris, Bourneville [2] made the following classification of idiocy, based upon researches during the past 25 years at La Salpêtrière and Bicêtre: (1) Idiocy symptomatic of chronic meningitis (meningitic idiocy). (2) Idiocy symptomatic of chronic meningo-encephalitis (meningo-encephalitic idiocy). (3) Idiocy symptomatic of arrested development of the convolutions without malformations, with lesions of the nerve-cells (congenital idiopathic idiocy). (4) Idiocy symptomatic of hypertrophic or tuberous sclerosis. (5) Idiocy symptomatic of atrophic sclerosis, (*a*) sclerosis of a hemisphere or of both hemispheres of the brain (hemispheric sclerosis); (*b*) sclerosis of a lobe of the brain (lobar sclerosis); (*c*) sclerosis of separate convolutions; (*d*) shagreen sclerosis of the brain. (6) Hemiplegic or diplegic idiocy, symptomatic of focal lesions due to vascular obliteration or to hemorrhage (pseudoporencephaly, etc.). (7) Idiocy symptomatic of simple ventricular hydrocephalus or complicated by extraventricular hydrocephalus (hydrocephalic idiocy). (8) Idiocy with pachydermic cachexia or myxedematous idiocy associated with absence of the thyroid gland. (9) Idiocy symptomatic of an arrested development of the brain with congenital malformations (true porencephaly), absence of corpus callosum, etc. (10) Idiocy symptomatic of microcephaly from arrest of develop-

[1] Brit. Med. Jour., Epitome, Sept. 22, 1900.

[2] Brit. Med. Jour., Epitome, Sept. 22, 1900.

ment with or without malformations, or having for cause lesions occurring after birth (microcephalic idiocy properly so-called or symptomatic).

Chas. H. Beard [1] gives the following description of the ophthalmoscopic picture presented by 2 cases of **amaurotic family idiocy**: The disc is of normal size and stands out unusually clear-cut as to its borders, and the choroidal ring is specially distinct: *i. e.*, free from the obscuring radiate striation normally present in the eyes of children. The lamina cribrosa is slightly veiled, though visible. Contrary to what he had been led to expect, there is not pronounced atrophy of the optic nerve and retina. The outer half of the disc is decidedly blanched, and the retinal vessels are all somewhat reduced in size. The choroid and the hexagonal pigment, wherever visible, appear normal. The great dominating feature of the picture, and that which characterizes this fundus and is absolutely diagnostic of the disease in question, is what one sees in and immediately around the macula lutea. Surrounding the fovea centralis, and concentric with it, though fully 2 or 3 times as large, is a liver-colored disc. This disc is the center of a zone of grayish-white, which extends for at least 2 discs' diameters horizontally, and somewhat less vertically in every direction from the center, and gradually fades away into the normal red-orange of the eye-ground. This livid disc is as clear-cut as a coin—not irregular in outline, as is the case in acute inflammatory conditions, where the surrounding retina is infiltrated; nor is it either cherry-red or carmine, as in those other instances, but is distinctly brownish. Instead of coinciding with the fovea in area, as is stated above, it is larger. That is to say, instead of marking the area which is occupied by the cones alone, it marks that which is devoid of the ganglion cells. Another highly distinguishing feature is observed in the character of the whitish zone surrounding the center. This is nebulous rather than cloudy. It is nearly white at the circumference of the liver-colored disc, thence gradually thins away to nothing, but is translucent and shows *some* color throughout. Far from obscuring the retinal vessels which enter it, it serves only to make them more distinct by contrast, so that one is able to trace the tiniest of them right up to the central spot.

**Periodic Psychoses.**—Marco [2] insists that **cross-markings on the nails** are of special frequency and regularity in the periodic psychoses. In cyclic insanity and other periodic mental disturbances the nails present grooves and ridges which enable one to estimate with some precision the frequency and relative duration of the attacks. In some cases in which the periods of mental disturbance are frequent, of short duration, and of regular occurrence, the nails assume an appearance resembling the striæ of the ridged shells of certain molluscs.

**Alcoholism.**—Cololian and Rodiet [3] found by studying a number of cases in which delirium tremens had been present or was threatened that hallucinations could be easily provoked by peripheral influences. For instance, inert substances brought in contact with the tongue would give rise to sensations of a distinct gustative variety. Such patients would insist that they tasted salt, quinin, strychnin, vinegar, or other well-known substances. In the same way stroking the mucous membrane over the turbinated bodies gave rise to hallucinations of the sense of smell, variously and graphically described by the patients. They observed that these phenomena do not apply solely to alcoholism, but are sometimes observed in neurasthenics and hysterics. The majority of patients lose this hyperexcitability in from 24 to 48 hours after their entrance to the hospital, but certain ones remain excitable for 10 or 12 days, and the persistence of the hallucinatory state seemed to be paralleled by evidences of degeneracy in given individuals.

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[1] Jour. Nerv. and Ment. Dis., 1900.

[2] Gaz. Med. Lombarda.

[3] Arch. de neurol., 1900.

**General Paresis.**—Ligui Mongeri,[1] basing his opinion on 47 cases, thinks that the disease is due to a complex of causes, of which the most important are syphilis, alcohol, and heredity, but that syphilis is necessary for the disease to develop; it is the factor which prepares the soil, while alcohol and heredity merely aid in its development. He asserts that without syphilis, congenital or acquired, it is impossible to have general paralysis.

**Classification of Mental Disorders.**—Kraepelin[2] gives the following classification: (I) Infectious psychoses: (*a*) febrile delirium; (*b*) infectious delirium; (*c*) states of infectious enfeeblement. (II) Psychosis of exhaustion: (*a*) delirium of collapse; (*b*) acute confusion; (*c*) chronic nervous exhaustion. (III) Intoxications: (*a*) acute; (*b*) chronic, including alcoholism, morphinism, cocainism, etc. (IV) Thyrogenic psychoses: (*a*) myxedematous psychoses: (*b*) cretinism. (V) Precocious dementia: (*a*) hebephrenic forms; (*b*) catatonic forms; (*c*) paranoiac forms. (VI) Paralytic dementia. (VII) Psychoses of cerebral lesions. (VIII) Psychoses of the period of involution: (*a*) melancholia; (*b*) delirium of presenile prejudice; (*c*) senile dementia. (IX) Depressive mania: (*a*) simple form; (*b*) periodic form; (*c*) circular form. (X) Systematized insanity. (XI) The neuroses: (*a*) epileptic psychoses; (*b*) hysteric psychoses; (*c*) traumatic neuroses. (XII) Psychopathic states: (*a*) constitutional depression; (*b*) obsessional insanity; (*c*) impulsive insanity; (*d*) sexual inversion. (XIII) Arrest of psychic development: (*a*) imbecility; (*b*) idiocy.

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[1] Rev. Speriment. di Fren., April 19, 1900.

[2] Rev. de Psychiatrie, April, 1900.

# DISEASES OF THE SKIN AND SYPHILIS.

By LOUIS A. DUHRING, M.D., and M. B. HARTZELL, M.D.,
OF PHILADELPHIA.

## INFLAMMATIONS.

**Urticaria.**—Philippson [1] does not believe that urticaria is due to reflex nervous action exerted on the blood-vessels, but holds with Heidenhain that a secretory action of the vascular endothelium is involved, and that the edema is similarly produced by direct action of poisonous substances upon the vessels in the neighborhood. He concludes that, just as in the case of the erythemas, urticaria is a mild inflammation, in which the irritant is of low intensity and exerts a more purely local action.

**Urticaria and Odors.**—Joal [2] reports 3 cases in which certain odors produced urticaria. In one the odors from aromatic essences used in the manufacture of liquors; in another, of iodoform; and in a third, the odor from roses, lilacs, and hyacinths, accompanied in the two last cases with symptoms of hay-fever.

**Caterpillar Rash.**—J. Cantlie [3] describes briefly several cases of this affection occurring in 3 children and 1 adult, the rash appearing suddenly. All the patients had just handled caterpillars that had been confined in a box. The caterpillars were of the "woolly-bear" variety. The rash was bright red in color, itchy, occupied the face and neck, and disappeared in 24 hours under simple remedies. A. M. Browne [4] states that the hairs of the "woolly-bear" caterpillar are armed throughout their length with a series of sharp spines, easily detachable from the hairs; these, when the caterpillar is handled, penetrate the skin and remain embedded when the hair itself is brushed away. The same is true of the "palmer-worm," the larva of the yellow-tail moth (Liparis auriflua), the exceedingly irritating properties of which are generally recognized.

**The Nature of Erythema Induratum.**—Thibierge and Ravaut [5] conclude, from a study of lesions taken from 3 cases of this affection, that it should be placed among the cutaneous tuberculoses, alongside of tuberculous gummata, with which it presents the greatest clinical affinities. Although tubercle bacilli were not found in sections of the lesions, the presence of vascular and inflammatory changes with giant cells was sufficient, in the opinion of the authors, to make probable the tuberculous nature of the malady. In a guinea-pig inoculated with a fragment taken from one of the ulcerating lesions a typical experimental tuberculosis was produced, thus establishing the tuberculous origin of the disease.

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[1] Giorn. Ital. delle Mal. Ven. e delle Pelle, 1899, Fasc. VI; Brit. Jour. of Derm., June, 1900.

[2] Rev. hebdom. de Laryng., June 10, 1899.

[3] Brit. Med. Jour., Aug. 29, 1899.

[4] Brit. Med. Jour., Sept. 23, 1899.

[5] Ann. de dermat. et de Syph., No. 6, 1899.

With the name of **"erytheme indure des scrofuleux of Bazin"** C. S. Dade[1] reports a case of this rather rare disease (with two photographs), and concludes, from the histologic and bacteriologic findings, that the disease is not entitled to be regarded as tuberculous, and thinks that it should be regarded as a simple subacute inflammatory manifestation, keeping the title "erythema induratum," but dropping "of the scrofulous." The disease in this case, as in many others that have been observed, had much in common with erythema nodosum, but nevertheless differed from it. [Audry[2] came to the same conclusion as Dade, that it was not scrofulous, but seemed to be rather a chronic form of erythema nodosum.]

**Dermatitis Venenata due to the Common Ivy.**—W. J. Munro[3] reports a case in which an inflammation of an erythematovesicular type was caused in a woman aged 40 by contact with the wet leaves of the common ivy (Hedera helix). Four distinct attacks occurred, each marked by an increased susceptibility to the poison. The lesions were grouped in places and bore a resemblance to herpes zoster.

**Chronic Primal Dermatitis.**—E. Heuss,[4] of Zürich, describes a case of chronic dermatitis due to contact with Primula obconica. A characteristic feature of dermatitis due to this plant is the suddenness of the attack, affecting usually the hands and face, with itching, swelling, redness, vesiculation, and papulation. The poisonous properties of the plant are due to fine hairs on the under surface of the leaves.

**An Epidemic of Impetigo Contagiosa.**—Ohmann-Dumesnil[5] reports 13 cases that were traced from one individual to another, showing contagion, with one exception all occurring in infants or children, and for the most part in several families. Antiseptic ointments of camphor and carbolic acid were successfully employed. Attention is called to the point that the diagnosis should be made early and treatment instituted before opportunity for spreading has been given.

**Contagious Impetigo.**—According to Abraham,[6] the pustular and crustate eruption characteristic of this disease is due to infection with Staphylococcus aureus and Staphylococcus albus, and he claims that complete cure can be effected in a week or two by the use of the following antiseptic lotion and ointment: ℞. Creolin, ½ dram; water, 10 ounces. The patients are instructed to use this lotion mixed with equal parts of hot water, and then to apply the following ointment: ℞. Ammoniated mercury, 40 grains; ointment of rose-water, 1 ounce. The creolin when mixed with water forms a milky fluid. It is an excellent antiseptic and it allays itching.

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[1] Jour. Cutan. and Genito-urin. Dis., July, 1899.

[2] Ann. de Dermat. et de Syph., 1898.

[3] Australas. Med. Gaz., Jan. 20, 1900.

[4] Monatsh. f. prakt. Derm., July 1, 1899.

[5] St. Louis Med. and Surg. Jour., June, 1900.

[6] Phila. Med. Jour., Dec. 30, 1899.

**Clinical and Bacteriologic Study of Impetigo.**—Sabourand[1] in a preceding memoir maintained that two separate diseases have hitherto been confused under the term impetigo, the impetigo contagiosa of Tilbury Fox and the impetigo of Bockhart being distinct affections. The former is essentially a vesicular affection; the latter, a pustular. In the article now under review Sabourand arrives at the following conclusions: The impetigo contagiosa of Fox, characterized by clear phlyctenules, which are soon replaced by yellowish crusts, is a contagious and autoinoculable disease, of which the specific microbe is a streptococcus. Ecthyma is due to the same microbe, and is the same disease modified by certain conditions. The lesions of impetigo contagiosa readily and frequently become secondarily invaded by Staphylococcus aureus, and then suppurate, though the condition of suppuration is not a part of the disease itself. Although these infections do not really cause the vesicle of impetigo contagiosa, they may give rise to the formation of other lesions than those of that disease. Cultures of vesicular impetigo ought to be made in a liquid medium. The mistake of regarding all impetigos as being due to a staphylococcus is due to the fact that the lesions of vesicular impetigo may be early infected by staphylococci. Sabourand believes that most previous observers have mistaken the staphylococcus as the causal agent of streptococcic impetigo, whereas it is only a secondary infection. On the other hand, Sabourand's recent work confirms the labors of Crocker (1881) and more recently (1899) of Unna. He agrees with Balzer and Griffon that the streptococcus of impetigo is the "streptococcus of Fehleisen," of erysipelas, etc.

**Pemphigus of the New-born.**—Bloch[2] distinguishes a benign and a malignant form, the latter resembling foliaceous pemphigus, and to some extent also Ritter's exfoliative dermatitis, being nearly always fatal. It represents a generalized infection, due in most cases to streptococci, the mode of infection, however, being often obscured. It is contagious and is often spread by the midwife. The article is based on 20 cases, studied clinically, anatomically, and bacteriologically.

**Acute Pemphigus in the Adult.**—Köhler[3] gives the following account, which seems to establish the existence of **acute pemphigus in the adult:** In a place near Jena 5 persons who lived in two neighboring houses, 4 children and an adult, were attacked with the disease. All recovered except one of the children. In a week after the sick child's admission the nurse who had taken care of it was affected with blebs, fever, and the usual constitutional symptoms. The first bleb appeared on her face, because (the author thinks) the dead child had caressed her face with its hands. Two days previously another Sister, who had washed the child's linen, was attacked in the same way. Seven individuals were affected, 3 of whom were adults.

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[1] Ann. de Dermat. et. de Syph., Mar., 1900.

[2] Arch. f. Kinderh., vol. XXVIII, 1900.

[3] Deut. Arch. f. klin. Med., LXII, 5, 6; Centralbl. f. innere Med., Nov. 4, 1899.

Staphylococci and a diplococcus were found in the contents of the dead child's blebs.

**Pemphigus of the Buccal Mucous Membrane.**—Wm. L. Baum [1] reports a case of this very rare affection. The patient, a man of 25, had an eruption of large blisters on the dorsum linguæ, no pain, and slight inflammatory areola. Three distinct attacks occurred at intervals of about 6 months. The latest attack gradually recovered under the application of a mercuric chlorid solution (1 : 3000). There was no history of specific disease.

**Herpes Zoster of the Finger.**—Suermonprez and Platel [2] say that this disease affecting the fingers is not so rare in this region as is generally supposed. The metacarpal or middle phalanges are usually affected, the thumb very rarely being attacked. The authors believe it to be due in every instance to a direct infection of the finger, traumatism being only of secondary importance in its etiology. It differs from herpes elsewhere in the absence in the beginning of local congestion, a large flat bleb being the first lesion that occurs.

**Treatment of Herpes Zoster by Picric Acid.**—"Le Presse Médicale" [3] discusses the results obtained by different observers in the application of picric acid to herpes zoster. Delebecque has obtained good results from the application of an aqueous solution, in the strength of 12 parts to 1000, applied freely by gauze compresses or absorbent cotton. The compresses wet with the solution were retained in position by a roller bandage, which was removed after some days. Thiery and Floquet have also treated herpes zoster with picric acid, but have employed ethereal or alcoholic solutions of greater strength, the alcoholic containing 10% of the acid and the ethereal 5%. These applications are followed by some pain, which, however, disappears quickly. Brocard has prepared a collodion application composed of the following: Picric acid, 75 grains; cannabin, 25 grains; alcohol, 2 drams; ether, 3 drams; elastic collodion, 4 drams. This application relieves both the violent pains and the itching which sometimes accompany zoster. It protects the part and prevents the possibility of secondary infection. The crusts which are formed by this application are a valuable protection to the new epidermis.

**Eosinophilia in Dermatitis Herpetiformis (Duhring).**—M. A. Brown and G. P. Dale [4] record the blood count in a case of this skin affection coinciding with a marked increase of the eosinophiles, which, normally about 0.5%, became increased to 29%. In 2 months this rose to 45%, but sank in a few weeks to 30%. The patient, a man of 50, at this time had suffered from dermatitis herpetiformis (Duhring) for 27 years, undergoing frequent attacks beginning with pruritus and developing the characteristic vesicular, sometimes bulbous, lesions, which came always on the hands and at times affected the feet and inner aspects of the thighs. Treatment was of little value, but spontaneous amelioration would occur from time to time. No history of syphilis nor of the diseases of childhood existed. The presence in the muscle-substance of the patient of Trichinæ eosinophiliæ was not established after careful examination, nor were any evidences of its former residence there found.

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[1] Phila. Med. Jour., June 9, 1900.

[2] Jour. des mal. cutan. et syph., Dec., 1899.

[3] Dec. 16, 1899.

[4] Jour. Am. Med. Assoc., Feb. 17, 1900.

**Dermatitis Herpetiformis; Type Hydroa Gestationis.**—A typical case is reported by J. Galloway,[1] emphasizing the well-known characteristics of (1) superficial inflammation; (2) tendency to multiformity of eruption in any attack and change of type in different attacks; (3) tendency to herpetiform grouping; (4) much disorder of sensation; (5) chronicity, relapses, and recurrences, all being common. The patient, a female aged 40, suffered at each confinement since her sixth and extending to the thirteenth, each succeeding attack becoming more severe and lasting longer. As to arsenic, it is of little use in the treatment, though in other forms of dermatitis herpetiformis arsenic is claimed to be valuable.

**Value of Arsenic in Dermatitis Herpetiformis.**—A physician residing in India [2] gives his personal experience, extending over several years, with this disease, and points out the striking amelioration of the disease during attacks of malarial fever and other intercurrent disorders, and the benefit derived from the use of arsenic in full doses.

**A Mucor Dermatitis Resembling Scabies.**—Emil Luck [3] describes a disease which had repeatedly been mistaken for scabies, having the general appearance of that disease, the seats of predilection also being those of scabies. There was violent itching. From continual scratching there existed marked excoriations. Sarcoptes scabiei could not be found, nor would the condition yield to the remedies ordinarily employed against that disease. In examining the contents of some of the highly inflamed pustules mycotic filaments were discovered, resembling mucor corymbifor. The affection did not yield to chrysarobin, ichthyol, or red precipitate ointments, but disappeared under the use of a 3% menthol and salol ointment.

**Treatment of Furunculosis by Galvanism.**—Gustav Langman,[4] recalling the well-established pathogenesis of carbuncles and furuncles, the presence of Staphylococcus pyogenes aureus, and the attendance of a trophoneurosis, insists that an experience of some 20 years has shown to him the slight value of ordinary treatments and the great benefit of galvanism in these conditions. Staphylococcus pyogenes aureus is thought to be a more or less constant inhabitant of the skin follicles. The occurrence of furuncles after wasting disease is indicative of some trophic alterations. In diabetes it is true that the condition of the blood may cause carbuncles; this is because sugar is an excellent culture-medium for this staphylococcus. But often cases occur in which the distribution of the lesions is along the course of some nerve tract. The application of galvanism to such cases affords relief quickly, the discharge, desiccation, and cure occurring in a few days. Incision is condemned. From 2 to 6 applications of the cathode will be sufficient to cure, and, in addition to this, a pledget of cotton soaked in bichlorid (1 : 1000) will hasten the result. The effect of this is cataphoric, anesthetic, electrolytic, trophic, and, in an incipient case, abortive to the process. In conclusion a list of authorities supporting this method is quoted.

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[1] Practitioner, May, 1900.

[2] Brit. Jour. Dermat., July, 1899.

[3] Med. Rec., Aug. 5, 1899.

[4] N. Y. Med. Jour., June 2, 1900.

**Salicylic Acid for Furunculosis.**—Philipson [1] advises the application of a plaster containing 50% of salicylic acid in the treatment of a well-formed boil. This should be changed 4 or 5 times a day, thereby hastening the necrotic process. When the core is eliminated, treatment which favors granulation may be instituted. Minute furuncles are arrested by the application 3 times a day of pure alcohol, or alcohol containing 5% of tincture of benzoin. Larger furuncles may be washed with alcohol containing 2% of salicylic acid. In generalized furunculosis the parts should receive a warm bath daily and then be rubbed with vaselin containing 2.5% of salicylic acid.

**Rapid Method of Healing Boil and Abscess Cavities.**—M. B. Hutchins [2] urges the employment of antiseptic poultices, in the form of flaxseed meal made with about 3% carbolized water and applied directly over the wound and cavity, or, preferably, with a layer of gauze intervening. By this means continuous drainage through absorption in the poultice is obtained, and all pus and necrotic tissue come away. The carbolic acid prevents reinfection, and the warmth and moisture prevent premature union of the cut edges.

**The Etiology of Ecthyma Gangrænosum.**—Hitschmann and Kreibich [3] report a third case of ecthyma gangrænosum. The patient was a very emaciated infant 6 weeks old. Over the buttocks, the thighs, and the abdomen, and in the popliteal spaces were from 25 to 30 lesions, varying in size from a lentil to a small coin, covered with necrotic detritus. Some of these lesions exhibited a central depression. Microscopic examination of the lesions revealed the presence of a bacillus, which proved to be Bacillus pyocyaneus. From the three cases studied by the authors they conclude that Bacillus pyocyaneus is the exciting cause of this affection, which occurs in distinctly cachectic children, and is characterized by the appearance of red-brown or dark-brown discolorations of the skin, usually surrounded by a halo. The centers of these efflorescences speedily become necrotic.

**Acne Urticata.**—Löwenbach [4] reports a case of this unusual affection, with a microscopic study of the lesions. A man of 35 had suffered 10 years from a very itchy eruption, which had resisted all forms of treatment, the parts affected being the extensor surfaces of both forearms, the axillas, the supraclavicular and infraclavicular regions, the neck, the scalp and forehead, along the spinal column, around the anus, the inguinal regions, and the popliteal spaces, but the parts which suffered most were the nape, the scalp, and the forehead. The eruption, which began as slightly reddened hard wheals, was preceded by intolerable burning and itching; with the appearance of the wheals the itching diminished somewhat, but was still severe. The wheals continued to enlarge for a short time peripherally; a drop of serum appeared in the center, which dried into a crust; after some days the center of the lesion became depressed, with diminution of the itching, and the crust fell off, leaving a shallow, depressed scar. As the old lesions disappeared new ones appeared, and in this manner the disease continued year after year. A microscopic examination showed an inflammatory edema of the cutis, which led to a collection of free fluid in the papillary body and the consequent formation of a small, epidermoidal vesicle upon the point of the papilla. In the center of the wheal was a necrotic mass, pierced by the duct of a sebaceous gland, the gland itself and the accompanying hair follicle being surrounded by an intense infiltration of polynuclear leukocytes. Clinically and microscopically acne urticata appears to stand midway between acne necrotica and chronic urticaria.

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[1] Deut. med. Woch., May 4, 1899.

[2] Atlanta Med. Jour., April, 1900.

[3] Arch. f. Dermat. u. Syph., Bd. L, Heft 1.

[4] Arch. f. Dermat. u. Syph., Bd. XLIX, Heft 1.

**Suppurative Perifolliculitis and Frambesiform Vegetations following Eczema.**—Huber [1] reports the following unusual complication of eczema: In a young girl, aged 17, who had suffered from a severe universal eczema for 4 years an eruption of pea-sized, firm, perifollicular papules occurred on the anterior surfaces of both thighs and the mons veneris. A similar eruption existed upon the flexor surface of the left elbow. Upon the posterior surface of the thighs were coin to palm-sized, elevated, partly ulcerating, proliferating patches, formed by the confluence of numerous perifollicular lesions, these patches being polycyclic or oval in shape. The elementary lesion was a pustular perifolliculitis; through the coalescence of the pustular lesions and their ulceration frambesiform vegetations arose. When left to themselves, the vegetations possessed unlimited powers of increase; but if treated antiseptically, they disappeared in a very brief period, leaving no trace. The author regards the complication as due to the invasion of pus-producing organisms in an individual predisposed thereto.

**Treatment of Scabies.**—S. Sherwell [2] describes his method of treatment as follows: Usually several members of a family are infected; each one is directed to take a bath the same evening, and adults should use a sand soap over the tougher portions of the body which are involved. The body and limbs are then rubbed lightly with a little sulphur lotum, about a half teaspoonful for each individual; no great amount of friction is required. The bed linen and underclothing should be changed, and between the sheets a little sulphur should be placed in each bed, and the sheet shaken, so that the sulphur is disseminated over the whole internal surface. By repeating the powdering of the bed, and by bathing and changing clothes in about the same way every other night for a week, the cure is effected in ordinary cases. An exaggerated case will, of course, take longer. One ounce of sulphur lotum is in excess of what is needed in the ordinary treatment of a family of 5. This treatment is more cleanly and less irritating than the other methods usually employed.

**A Case of Chronic Interstitial Nephritis in which Dermatitis**

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[1] Arch. f. Dermat. u. Syph., Bd. XLIX, Heft 1.

[2] Jour. Cutan. and Genito-Urin. Dis., 1899.

**Exfoliativa Supervened.**—Duckworth [1] reports the case of a woman, aged 59, who, after having been ill 5 months, developed a rash all over the body "like measles." This disappeared in 2 days' time, and was followed by desquamation. Two or 3 months later she suffered from a universal dermatitis, the skin being dry and harsh, and covered with thin, white scales lying on an erythematous base. Behind the ears were some small moist eczematous patches, and the scalp was covered with fine scales. The entire surface was tender to pressure. The eyelids were puffy, and there was edema of the feet and legs. The urine was acid and contained albumin. After a time the patient became drowsy, the urine scanty, and death from uremia followed. The author regards the appearance of albuminuria in the exfoliative dermatitis of elderly people as an unfavorable factor, and the occurrence of ordinary eczema over large areas in elderly subjects with chronic nephritis is of evil omen. Inunction with pure olive oil is the best local treatment.

**Buckwheat Flour in Acute Eczema.**—B. C. Patterson [2] says that buckwheat flour will be found most useful for acute eczemas and for poultices. For eczema in the wet stage the application of 1 part of zinc oxid to 12 or 18 parts of buckwheat flour is recommended. A poultice of this flour, with a little carron oil added, is regarded as a most soothing application both for inflamed ulcers and for irritable eczema.

**Chronic Eczema Treated by Naftalan.**—Rosenbaum [3] gives a résumé of his experiences with naftalan, especially in burns and in acute and chronic eczema. The results obtained in chronic eczema were more striking than those in acute eczema. One man had suffered for more than 20 years from an obstinate fissured and crusted eczema of both hands. In a short time after the naftalan treatment was begun the itching ceased, and the skin rapidly took on a normal appearance. In another patient, a woman, there was an eczema which extended over almost the entire surface of the body, so that the appearance was similar to that of ichthyosis. In 40 days after the beginning of the treatment with naftalan she was completely cured.

**Treatment of Eczema by Picric Acid.**—Francesco Radaeli [4] reports upon the use of this substance in 10% aqueous solution upon 25 patients. There was not noted any yellow coloration of the conjunctiva nor changes in pulse-rate or temperature. The urine, however, was diminished in amount and of a yellowish-red color, although no picric acid could be detected in it. In 14 of these patients suffering from acute eczema the results were satisfactory in a short time. Of the other 11, four showed a marked benefit after application of picric acid used alone, or later alternating with other remedies. In 7 instances there was no benefit. Some of these were benefited by moist applications of salicylic acid, while others resisted all methods. In a few instances applications of picric acid seemed to aggravate the local conditions, increasing the congestion, the edema, and the number of vesicles. The following conclusions are offered: (1) This method should be tried in acute eczema and in the exacerbations of chronic eczema. (2) When well borne, and it generally is, after a brief period of smarting the symptoms are relieved. (3) This betterment generally goes on to cure; rarely the benefit is temporary. (4) If not well borne, it does not cause severe local disturbance. (5) It appears to act as an antiseptic and keratoplastic agent.

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[1] Brit. Jour. of Dermat., Jan., 1900.

[2] China Med. Missionary Jour., Jan., 1900.

[3] Therapist, July 15, 1899.

[4] La Settim. Med., No. 9, p. 97, 1899.

**Sodium Cacodylate in Psoriasis.**—Neumann,[1] at a séance of the Vienna Dermatological Society, exhibited 2 patients with psoriasis who had been treated by injections of sodium cacodylate. The first patient, a girl of 19, who had not been treated before her entrance into the hospital, received at first injections of a solution 2 : 20; later the strength of the solution was increased to 4 : 20, and then to 6 : 20. After 20 injections the eruption, which was very extensive, began to undergo involution, and after 30 the scales fell off, leaving smooth red spots. Although the amount of sodium cacodylate seemed large, no unfavorable effects were produced. The second patient was also a young woman, aged 20, with an extensive eruption. She received daily injections of a 20% solution.

**Eczema Treated by X-Rays.**—Mackey [2] gives the notes of 2 cases of eczema treated by the x-ray. The first case was a boy, aged 11, who had been repeatedly treated in the usual manner without marked benefit, the parts affected being the legs, arms, and cheeks. After treatment with lotions and salves for nearly a month, without improvement, the x-rays were used for 10 minutes daily, at 4 inches' distance. After a month of this treatment marked improvement was noted, the case being almost cured. The second case was a girl, aged 13, who had had an eczema continuously for 2 years, the parts affected being the bends of the elbows and the region about the mouth. After 16 applications of the x-rays, made as in the first case, the right arm was practically well, but the left one was still eczematous. It is important that the treatment be suspended if signs of irritation appeared. Holland [3] records the case of a woman of 19 who presented a chronic eczematous eruption covering the dorsum of one hand, following the evacuation of an abscess that had formed over a metacarpal bone, the skin being thickened, scaly, and cracked, and the seat of adherent blackened crusts. After 7 exposures at intervals of 4 or 5 days to the action of an x-ray tube at a distance of from 4 to 7 inches, and for periods of 15 minutes, the eczema disappeared.

**Treatment of Psoriasis Vulgaris.**—Unna [4] succeeded in curing an obstinate case of psoriasis that resisted all treatment, by the use of the following salve: ℞. Acidi salicyl., acidi pyrogallici, of each, gr. xlvi; ammon. sulph. ichthyol, olei oliv., of each, ʒiiss; adeps lanæ, ℥iv. By the aid of the salicylic acid and of the ichthyol the effect of the pyrogallic acid was greatly enhanced. In this form, it is stated, the salve may be safely applied also to the face without producing any irritation or discoloration of the skin.

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[1] Arch. f. Dermat. u. Syph., Bd. XLIX, Heft 1.

[2] Brit. Jour. of Dermat., April, 1899. [3] Brit. Med. Jour., April 29, 1899, p. 1024.

[4] Boston M. and S. Jour., Nov. 16, 1899.

**Psoriasis Vulgaris in the Infant.**—J. H. Rille[1] reports a case occurring in an infant 5 days old. He furthermore calls attention to the fact that psoriasis in children often follows one or another of the acute eruptive fevers and vaccination. Cases in which the disease occurred at an unusually early age have been reported by Billard (3 months), Zeissl (8 months), and Neumann (4 months).

**Treatment of Psoriasis by Intravenous Injections of Arsenic.**—Karl Herxheimer[2] treated 28 cases of psoriasis with intravenous injections of arsenic, and obtained cures even in cases in which no local treatment was applied. The cure was usually evident by the end of the second or the beginning of the third week. The average duration of the treatment was about 48 days. The initial dose of the arsenious acid was $\frac{1}{60}$ of a grain, which was increased daily by $\frac{1}{60}$ until $\frac{1}{10}$ of a grain was reached, this last remaining until the final disappearance of the efflorescence. The injections were mostly made into the elbow-joints, the forearm being compressed with an Esmarch bandage, which made the veins very prominent. To prevent any recurrence, the author recommends intermittent treatment, similar to what is done in syphilis during the periods free from symptoms.

**Blastomycetic Dermatitis.**—Hyde, Hektoen, and Bevan[3] report a case of this rare disease. It is due solely to the invasion of the skin by one of the plant forms of the yeast family. In its clinical aspect it resembles lupus vulgaris in the ulcerative stage. Potassium iodid is worthy of a trial in all cases.

**A Case of Blastomycetic Dermatitis Engrafted on Syphilitic Ulcers.**—Anthony and Herzog[4] report the following case: A man, aged 44, presented numerous ulcerative lesions upon the foot and leg, many of them being covered with fungous granulations projecting a considerable distance above the surface of the sound skin. The ulcers followed an erysipelas of the leg, which had occurred some 20 years previously, and from the time of their first appearance the leg had never been freed from ulceration. A diagnosis of syphilis was made; but, with a view to investigating the possibility of the vegetating character of the ulcers being due to a subsequent blastomycetic infection, a microscopic examination of some of the lesions was made. The histologic changes found corresponded to those described as characteristic of blastomycetic dermatitis, and numerous blastomycetes were found.

**The Rôle of the Staphylococcus in Skin Diseases.**—C. T. White[5] discusses elaborately the importance in skin diseases of this pyogenic micro-organism. He marks the relative frequency of suppuration secondarily as double that occurring primarily, and quotes Wickham, who asserts that impetigo, sycosis non-tricophytica, paronychia, ecthyma, furuncle, carbuncle, etc., are sister maladies and have a common etiology, the direct causation being the action of the staphylococcus.

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[1] Jour. des mal. cutan. et de syph., July, 1899.

[2] Boston M. and S. Jour., July 27, 1899.

[3] Brit. Jour. of Dermat., July, 1899.

[4] Jour. Cutan. and Genito-urin. Dis., Jan., 1900.

[5] Boston M. and S. Jour., Sept. 7, 1899.

The presence on the scalp, and elsewhere on the skin generally, of this micro-organism enables it to effect entrance when abrasions occur and the hygiene of the individual is improperly cared for. Hence among the lower classes, where pediculosis with its resulting scratching is common, these suppurating diseases appear often and severely. Inoculation of pediculi will produce many of the above-named diseases, and self-inoculation is possible, since the germs are resident under the finger-nails. Correction of hygienic errors, and the use of 5% boric acid solution in children, after pediculi, etc., have been removed by appropriate treatment, will meet the indications.

**Pityriasis Versicolor.**—C. W. Allen[1] reports several cases of this disease occurring upon the face. He gives the following test: When Lugol's solution is painted on a patch of this affection a darker tint is produced than if the tissue were normal or some other disease present.

**Tinea Versicolor on the Face.**—A. Powell,[2] of Cachar, East Bengal, India, contrary to European and American observation, finds tinea versicolor occurring on the face common in that country. In 3 months he saw 56 persons thus affected, in 9 of whom the diagnosis was confirmed by the microscope. Specimens of the fungus and scrapings from the skin were sent to the London Dermatological Society, and were there examined by some of that Society's members, who pronounced the fungus to correspond to that observed in England. On black skins the patches of disease show as light patches of a yellowish color. As the author remarks, "'East is east and west is west,' . . . and miracles of Kew are plain facts at Khatmanda."

## HYPERTROPHIES AND ATROPHIES.

**Chrysarobin as a Specific for Warts.**—G. W. Fitts[3] says that warts upon the feet are to be distinguished from corns by the fact that they appear upon the plantar surface and that they bleed from a vertical bundle of papillas somewhat above the pared surface. Warts in this situation may be mistaken for corns, but the treatment by applications of salicylic acid and collodion, which is so efficient in the removal of corns, has no effect upon the wart. Chrysarobin is applied in 10% solution dissolved in ether or gutta-percha. The application should be made once a day, and the wart should be pared each time it is used.

**The Treatment of Warts.**—Du Castel[4] believes that warts are due to a specific bacterium and are very autoinoculable; but that they may readily be destroyed by all caustics, applied directly either to the surface of the wart or to the wound left after its avulsion. Nitric acid is placed first in the long list of cauterants. Local anesthesia is satisfactorily accomplished by ethyl chlorid. The galvanocautery or thermocautery is an excellent instrument for the total destruction of these growths. Kaposi recommends the following pastes as especially serviceable for the treatment of warts on the back of the hands and on the face: Flowers of sulphur, 20 parts; glycerin, 50 parts; diluted acetic acid, 10 parts. A 10% salicylate plaster is efficacious for the treatment of small warts; to this may be added resorcin. The plaster should be removed every 2 or 3 days, the wart first being scraped. A common form of treatment consists in the use of salicylic collodion, made up of salicylic acid 1 part and flexible collodion 8 parts. This is painted over the surface of the wart, and the application renewed every time the pellicle thus formed is rubbed off. Sublimate collodion is sometimes used, but it is extremely irritating. It is made of: Sublimate, 0.2 parts; collodion, 10 parts. The smooth, flat warts of children require less severe application than the rough, hard warts of adults. The seborrheic warts of the aged, made up of an epithelial layer placed on a papillary overgrowth, become so frequently the starting-points of cancer that they should be treated promptly.

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[1] Phila. Med. Jour., June 9, 1900.

[2] Brit. Jour. of Dermat., April, 1900.

[3] Boston M. and S. Jour., June 29, 1899.

[4] Jour. de Praticiens, No. 34, 1899.

**The Treatment of Scleroderma by Thiosinamin.**—Hebra,[1] at a meeting of the Dermatological Society of Vienna, presented 3 cases of scleroderma which had been treated by hypodermic injections of thiosinamin. The first patient had received 24 injections, and the skin, which in the beginning was tense and shining, had become approximately normal. In the second patient, after 24 injections, the hard circumscribed area diminished in extent and resistance. In the third patient improvement was manifest after the fourth injection. Half a Pravaz syringeful of 15% alcoholic solution was injected every second day in the interscapular region, the injections being made deeply, in order to avoid the production of superficial necrosis of the corium.

**Scleroderma; Its Treatment by Arsenic.**—L. Lindemann,[2] in view of the bad prognosis in this disease,—quoting Lewin and Heller, who assert that out of 203 cases on record but 16 were cured, and but 30 others at all improved by treatment,—thinks the arsenical therapy worth notice, especially since a case is presented in which undoubted gain occurred; the patient's general health improved and weight was added; the spread of the disease was completely checked; some patches were even diminished in size and lessened in abnormal appearance. The treatment by arsenic consisted in daily, later morning and evening, hypodermic administrations of *arsenious acid*. The solution was of such strength that 0.2 cc. of it contained 0.002 cc. of arsenious acid. Beginning with 0.2 cc. solution, this was increased each day until 0.9 cc. had been given; then it was started again at 0.1 cc. According to the symptoms, the dose should vary. If cardiac or psychic depression occur, it is well to refrain from the injection for a day or so. The duration of treatment in this case was 3 months, and when stopped the patient was suffering from malaise and pains, especially marked between the shoulders. The history of this patient, a woman of 50, is given in detail.

**Nitroglycerin in Xeroderma.**—B. Basket[3] reports the case of a boy aged 9 affected from the age of 2 months with xeroderma that improved markedly under the use of very small doses of nitroglycerin, taken 3 times daily. In 14 days an improvement was noted. The drug was used for 3 months, with the result that the former xeroderma had given way to a dry, eczematous-looking condition, the skin being soft, supple, and moist.

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[1] Arch. f. Dermat. u. Syph., Bd. XLVIII, Heft 1.

[2] Deut. Arch. f. klin. Med., vol. LXVI, Fest. XXV.

[3] Brit. Med. Jour., Nov. 4, 1899.

**Acanthosis Nigricans.**—T. Burmeister,[1] from a study of 1 case under his own observation and of 18 cases found in medical literature, endeavors to obtain the average sex and age of persons attacked by the disease, the duration, and the special localization of the cutaneous manifestations. Neither alcoholism, syphilis, nor tuberculosis was found to be an etiologic factor. The theory regarded with favor is that in acanthosis nigricans the function of the abdominal portion of the sympathetic nervous system is altered by a carcinomatous process giving rise to the disease of the skin and mucous membrane. The autopsy performed in the case under the author's observation did not, however, reveal any signs of a tumor or of a diffuse carcinomatosis. The nervous systems, central or sympathetic, could not be examined.

**Arsenical Pigmentation and Keratosis.**—L. P. Hamburger[2] gives a case in which a man aged 42 took liquor potassii arsenitis, from 5 to 8 minims each dose, for a period of 8 years in order to cure psoriasis. The use of the arsenic was followed by extensive pigmentation and keratosis, the latter especially on the palms and soles. The pigmentation was general, and was not always characteristic enough to be distinguished from the bronzing of Addison's disease. The author reviews the literature of the subject, and concludes that patients should never be permitted to use arsenic for a long period except under the observation of the physician.

**Destruction of Superfluous Hair by the X-Rays.**—James Startin[3] reports the case of a woman, aged 33, who consulted him for a growth on each side of the chin of about 30 dark hairs, about from ½ to ¾ of an inch long. Seven exposures of the x-rays were given, with the result that she had slight dermatitis, which got well in about a fortnight. All the hairs disappeared and showed no signs of return. Another case was that of a young girl, aged 23, with numerous dark hairs, which had grown freely on both sides of the face; after 18 exposures there was slight dermatitis, and in a month after the last exposure the hairs had completely disappeared. Two other women since, one 25 and one 27 years of age, had 6 and 7 exposures successively, and the skin showed slight electric dermatitis, with the same successful results. The roots and sheath, when examined microscopically, show a drying up of the hair bulb and root sheath.

**Hydrogen Dioxid in Hirsuties.**—L. Duncan Bulkley[4] says that the *bleaching* properties of hydrogen dioxid have long been used for changing the color of the hair, and advantage may be taken of this in connection with the growth of superfluous hair on the face of women. In a very considerable number of cases he has employed it when the hair was too fine to admit of its removal by electrolysis, and yet was very perceptible and troublesome. Especially on the upper lips of girls this condition is often the source of much distress. Here the free and repeated use of hydrogen dioxid will produce a very material improvement in the appearance in a very short time. By blanching the hairs a mustache which was very striking will hardly be noticeable at a short distance. But another advantage in the use of the drug in hirsuties, which, he believes, has not been heretofore mentioned, is a certain retarding influence which it exerts on the growth of the hair. This he has noticed in a number of instances for the past 2 or 3 years. This result is slow, but with a faithful continuance of the remedy the fine growth of hairs certainly diminishes. In applying the drug to such cases it is often well to begin by diluting it one-half with water, and increasing the strength gradually, for when a strong article is applied to the healthy skin, it will sometimes cause desquamation.

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[1] Arch. f. Dermat. u. Syph., vol. XLVII, p. 943, 1899.

[2] Johns Hopkins Hosp. Bull., April, 1900.

[3] Lancet, Mar. 3, 1900.

[4] Jour. Am. Med. Assoc., Dec. 23, 1899.

**Loss of Hair.**—George T. Jackson[1] gives an exhaustive tabulation of some most valuable and interesting statistics covering over 300 cases in which the various clinical facts are contrasted and analyzed. The past histories and occupations of the patients give rise to some conclusions which can not all be given here on account of space, but which are extremely *à propos*, the most important being: The loss of hair is more common in men than in women, possibly because women value their hair more highly and care for it better. Marriage has no direct bearing. The intellectual classes suffer far more; by these classes is meant: actors, architects, bankers, brokers, housekeepers, lawyers, manufacturers, merchants, nurses, physicians, students, teachers, and telegraphers. Of the cases, 60% begin at an age below 30. In women the trouble begins as a general thinning, but in men the whole top of the head suffers; this may have some relation to the fact that women preserve the infantile fatty cushion under the scalp much longer than men. Heredity is the predominate predisposing cause. Complicating diseases are usually those which affect the general nutrition. Dandruff is the most usual exciting cause; this includes pityriasis and seborrhœa sicca. Dandruff may be present without great loss of hair, however, but it is a more frequent cause than heredity, debilitating disease, keratosis, hyperhidrosis, or atrophic processes of the scalp in general. As to treatment, a 10% mixture of precipitated sulphur in good cold-cream, with or without 3% salicylic acid or extract of jaborandi (ʒj–℥j), frequently produces amelioration; resorcin, or an ointment of ammoniated mercury, gr. xx, calomel, gr. xl, in an ounce of vaselin, has been of use in some cases. Further, the use of massage, in cases without dandruff or in which that condition has been relieved, will be found very beneficial. With this, a lotion of resorcin, at first 3%, later increased gradually to 10%, used night and morning, may be prescribed. The sulphur cream is applied twice a week, and is less trouble than the lotion. The prognosis is bad when calvities is already present. An unfavorable family history as to the hair is significant. The longer the dandruff has lasted, the worse is the prognosis. It is less easy to help a man than a woman.

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[1] Med. Rec., May 26, 1900.

**Two Epidemics of Alopecia Areata in an Asylum for Girls.**—Bowen[1] reports 2 epidemics of alopecia areata in an institution for homeless girls between the ages of three and fourteen years. The first case occurred in a girl of 11, who when first seen presented 3 round, bald patches upon the crown of the head, clinically typical of alopecia areata. Several weeks later another girl was found to have a bald patch upon the crown, which increased rapidly in size for a time. Four months after the discovery of the first case a large number of the girls in the asylum were found to be affected. After cutting the hair of all the children, it was found that 63 of the 69 girls had bald areas upon the scalp. One girl, who had just entered the institution, acquired a patch in 3 days. After 2 months the disease appeared to come to a standstill; at the end of 6 months almost all the bald patches were covered with hair. No trace of micro-organism was found. No adult inmate of the asylum was attacked. Six years after this epidemic the first patient, having been absent from the asylum for 3 years, was readmitted. A month or 6 weeks afterward one of the children was discovered to have a bald patch, and 4 or 5 months later 26 of 45 children were affected in a similar manner.

**Lactic Acid in Alopecia Areata.**—Balzer[2] recommends the use of lactic acid, with water or alcohol, equal parts, in the form of a topical application made with friction. If the irritation is excessive, the strength is to be reduced or the treatment suspended for several days. [The solution should be made weaker at first, for lactic acid is somewhat caustic and should be employed cautiously.]

**Diseases of the Nails, with Special Reference to Their Significance as Symptoms.**—Jonathan Hutchinson[3] gives expression to the following views: "It is a well-known fact that any severe illness is liable to leave transverse furrows across the nails; that these furrows will always be found to be more marked in the thumb-nail than in any other, the forefinger next frequently, and that they shade off as you pass from the thumb to the little finger; that in some cases the arrest of nutrition, which is the cause of this temporary change in the nail, produces not a depression, but a white line on the nail. In the cases in which the vertical lines are produced some disturbance of nutrition appears to take place at the root of the nail, in connection with the general health. A great many cases of psoriasis occur as psoriasis of the nails without any lesion of the body. There are cases in which the nail becomes loose in its bed in healthy persons, many nails being affected at the same time, without any other obvious cause than that very often there is psoriasis in some relative. In the condition of the nail which goes with eczema there is generally a longitudinal furrowing of the nails, or the development of little indentations, as though the nail had been pin-pricked. Irregularity of the surface of the nail, beginning at its root, is typical of all eczematous conditions; loosening of the nail, beginning at its free edge, is characteristic of psoriatic conditions. In acromegaly, in which the digits themselves are very much enlarged, it happens that the nails do not enlarge at all; whereas in the false form of acromegaly, which occurs in connection with thoracic disease, either chronic or obstructive, in which the digits become enlarged and the fingers clubbed, the nails become of enormous size. A condition of great thickening and hardening of the nails sometimes occurs in connection with psoriasis and sometimes in connection with syphilis."

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[1] Jour. Cutan. and Genito-urin. Dis., Sept., 1899.

[2] Gaz. hebdom. de méd. et de chir., Jan. 18, 1900.

[3] Med. Age, Oct. 25, 1899.

## NEOPLASMS.

**The Treatment of Keloid by Static Electricity.**—J. Bécue [1] describes 6 cases of keloid treated by static electricity, a method introduced by Derville, who employed it to relieve the pain in a case of keloid over the sternum before scarifying it. Although the keloid measured 6 cm. by 2 cm., yet it disappeared after 3 applications. This was the most rapid cure, but the others were equally benefited, though less rapidly. The static electricity is employed in the form of sparks, and, according to the author, it gives the best results, at the same time relieving the pain. It is not painful, and acts rapidly. Each application lasts usually 8 or 10 minutes, but in the case of very small keloids it should not exceed 5 or 6 minutes, so that the operator may avoid forming vesicles. The applications should not be more frequent than once a fortnight.

**Keloid Treated by Electrolysis.**—Crocker [2] exhibited before the Dermatological Society of Great Britain and Ireland a female patient with keloid between the scapulas, which had followed a burn 5 months previously. The growth was very prominent, of a bright red color, and was accompanied by much itching and slight pain. After the employment of electrolysis at 4 sittings a fortnight apart, the growth was much shrunken and paler, and the itching and discomfort disappeared. The needle was inserted for a minute at a time, the punctures being about ¼ of an inch apart, and the current strength used was about 5 ma.

**Lupus Erythematosus.**—Karl Kopp [3] discusses very elaborately the nature of this disease and questions its relation to tuberculosis. It is stated that in all its occurrence, in causation, in situation, a close connection with tuberculosis is observed, and the microscopic appearance of the diseased tissue bears this out; so also does the test of animal inoculation, and the influence of corresponding therapeusis confirms this opinion. Various cases are quoted in substantiation of this. However, the conclusion is drawn with hesitation, owing to insufficient evidence for or against the positive relation of this disease to tuberculosis.

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[1] Paris Thesis, July, 1899; Treatment, Dec. 28, 1899.

[2] Brit. Jour. of Dermat., July, 1899.

[3] Deut. Arch. f. klin. Med., vol. LXVI, 1899.

**Treatment of Lupus Erythematosus.**—Hebra [1] states that he has treated successfully 6 cases of this disease with the external application of alcohol. By the aid of medicated cotton the alcohol is brought into contact with the affected region, without, however, exerting any pressure thereon, but simply by moistening it with the saturated cotton. After several moistenings the cotton is removed until the alcohol is evaporated, and then the process is begun anew. The more frequent and the more thorough the application, the sooner does the cure ensue. To enhance the antiphlogistic procedure, Hebra resorted lately to the following mixture: Absolute alcohol, sulphuric ether, and spirit of peppermint, in equal parts. Several cases have already been cured, without any recurrence as yet. Herman Lawrence [2] reports successful results by the following method of scarification: Use a fine-bladed scarifier (blades $\frac{1}{20}$ inch apart) under anesthetic, dust with iodoform and dress with zinc-glycogelatin, then apply a piece of solid india-rubber tubing over the dressing, to which pressure is made by strips of plaster, the pressure being continued for a considerable time. He has used this method also in cases of keloid, chronic eczematous patches, and other old-standing irritable skin inflammations.

**Treatment of Lupus Erythematosus.**—J. Bukovsky,[3] in considering its treatment, remarks that as long as its causation remains obscure, therapeusis must be empirical. Bulkley is quoted as finding it an angioneurosis, and as giving phosphorus. Other internal remedies have been recommended, but with none of the good results of local treatment. The application must be easily employed and comfortable, and the normal tissue surrounding must not be injured. Further, to be effective, it must be speedy and prevent extension into the unaffected region. Among such remedies are the following: (1) Soaps. These may have added various lead salts or sulphuric ether. Quicksilver plaster mulls and salicylic or resorcin soaps are of value. (2) Formulas of the following kind, such as have been suggested by Duhring: ℞. Zinc sulphat., potass. sulph., āā 20; aq. rosar., 120; alcohol, 15, may be found useful. (3) Lead-water with an addition of glycerin is the method used by Payne. (4) Iodin in various forms. (5) Naphthol. (6) Ichthyol. (7) Resorcin. (8) Pyrogallic acid. (9) Quicksilver plasters receive much praise from Hebra, Kaposi, Busch, Veiel, and others. (10) Peruvian balsam to which cod-liver oil is added is spoken of by Hebra, but this is rarely effectual. (11) Caustic alkalies; these are of wide-spread use. (12) Nitrate of silver, ammonium chlorate, and caustic soda are of value. (13) Acids are very useful, but must be used only for the following indications: When all other methods fail, when the disease is not wide-spread, and when such acids are chosen as do not eat too deeply. (14) In some cases surgical interference is justified. Various forms of scarification and of puncture are recommended. Three new methods have been advised recently in addition to these older ones: (1) Brocq and Veiel speak of the use of trichloracetic acid. Out of 11 cases 5 recovered, 2 improved, and 4 were negative. (2) Arsenic according to the method of Schuetz is used in a weak aqueous solution made from liq. potass. arsenit. with water added or containing, in addition, 1 or 2 drops of chloroform. Immediate beneficial results have followed. Sufficient data can not yet be obtained to realize its worth. (3) Brooke (Manchester, England) announces the uses of a salicylic-pyrogallic-collodion as follows: ℞. Ac. salicylic., 40; ac. pyrogallic., 10; collod., 100; M. Its use is simple, being applied from day to day according to effect. From what is known, this method is excellent, since the reaction is good, cure proceeds quickly, and pain is at a minimum. At the end of the article is noted a still more recent method, that of Hebra, consisting in applications of pure alcohol, which is said to be effective.

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[1] Boston M. and S. Jour., Nov. 16, 1899.

[2] Quarterly Med. Jour., Nov., 1899.

[3] Wien. med. Woch., No. 31, July, 1899.

**Lupus Vorax Cured by Local Applications of Guaiacol.**—Leplat[1] reports 2 cases with lupus vorax of the face treated by curetting and the subsequent use of bandages soaked in a solution of equal parts of guaiacol and glycerin. In one case the cure took place in 1 month; in the other, in 4 months.

**Treatment of Lupus by the X-ray.**—R. E. Scholefield[2] reports a case of lupus the successful treatment of which by this method is interesting in view of the unsatisfactory results of surgical interference. The patient, a male, aged 18, having suffered over a year and a half from a progressive lesion of the skin of the nose, during which various ointments, powders, and douches were tried, and even scraping resorted to, found that nevertheless the tip of the nose, the alæ, and the bridge were being destroyed. At this time the patient suffered from profuse night-sweats. The sole treatment then was poulticing. Wm. Webster, F.R.C.S., undertook the management of exposing the patient to the x-ray, which was as follows: Employing a Jackson high vacuum tube attached to a Newton-Apps coil giving an 18-inch spark, and using a hammer-break of the ordinary kind, the so-called "sun effect," described by Webster, was sought. The face, other than the affected area, was protected by a rubber mask of some thickness, and exposed to the rays for 10 minutes at a sitting at about 5 or 6 inches from the tube. This was continued every other day for a month, with the result that the skin dried up, scaled, and peeled off; the lupoid area where thickest forming a scab which, gradually becoming nodulated and pinkish in color, diminished and finally completely disappeared. The night-sweats stopped during this time. The final sitting occurred 5 months after the beginning of the treatment, and on the lungs being examined no signs of infection were discoverable; they were not looked for earlier. The author goes on to suggest that possibly these rays are the same as the photochemical rays of the solar spectrum, the work of Finsen's phototherapy giving very similar results. The penetrative power of the x-ray is insisted on as important, since, as night-sweats are so much relieved, the effect must be radical, and the fresh-air treatment for phthisis may be well supplemented by that of concentrated sunlight or exposure to the x-ray.

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[1] Ann. de Dermat. et de Syph., June, 1900.

[2] Brit. Med. Jour., May 5, 1900.

J. F. Hall-Edwards[1] says that the essentials of the method employed by him are exposure to a highly charged Crookes' tube for a considerable time at short distance. The average duration was 15 minutes and the distance from 1 to 2 inches. Three cases are quoted: (1) A case of lupus vulgaris on the instep: There were 5 exposures at intervals of from 2 to 3 days, except on the last occasion, when 9 days intervened. After the fourth exposure sloughing of the patch began and extended for about ½ of an inch around it. The resulting ulcer healed slowly with a sound scar. Two small adjacent patches of lupus which had been protected from the rays by sheet-lead ⅛ of an inch thick also became ulcerated and healed in like manner. (2) A case of lupus erythematosus on the arm: This patch was exposed on 2 successive days. It had sloughed by the twelfth day after the first exposure; a week later it was granulating, and ultimately healed well. C. T. Holland[2] gives the case of a boy of 16 who had suffered from lupus for 5 years and who was treated with rays from a vacuum tube with success. There were 17 sittings within 3 months. The hair on the same side of the head was lost, but grew again. Albers-Schönberg[3] reports the successful use of the x-rays in the treatment of 9 cases of lupus. The applications lasted about half an hour daily, and were continued until redness appeared. The skin first shows a slight yellow tint, soon followed by redness, which gradually deepens. Itching and pricking sensations, together with a feeling of warmth, are sometimes felt. Excoriation usually follows, giving the appearance of a burn. To protect the normal skin a mask of cardboard covered with tinfoil is recommended as better than lead-protection.

**Notes of Thought on Malignant Tumors.**—B. H. Buxton[4] discusses this subject, referring to current ideas, and draws the following conclusions: (1) Carcinomas contain connective tissue because there is an attempt at defense on the part of the organism. (2) Their increase is not prevented because the cells can multiply in the newly formed lymph-spaces of the stroma. (3) Sarcomas, as a rule, contain no connective-tissue stroma because they consist of the very few cells which would be called upon to form it, and which are now in revolt. (4) Sarcomas contain numerous blood-vessels. These always penetrate masses of immature connective-tissue cells, whether in the embryo or the adult. (5) Certain sarcomas contain connective-tissue stroma because the cells composing them are not ordinary connective-tissue cells, so that the latter are able to attempt defense. The defense fails for the same reason as it fails in the carcinomas. (6) Tumors containing connective-tissue stroma form metastasis via lymphatics because the newly formed lymph-spaces in which their cells are growing open up communications with the regular lymphatic channels. (7) Tumors containing no connective-tissue stroma form metastasis via the blood-vessels because the cells can easily break through the immature walls, while lymph-spaces are absent.

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[1] Edinb. Med. Jour., Feb., 1900.

[2] Liverpool Med.-Chir. Jour., Jan., 1899.

[3] Arch. of Röntgen-Ray, Nov., 1898.

[4] Jour. Cutan. and Genito-Urin. Dis., April, 1900.

**Treatment of Skin-cancer.**—A. G. Wollenmann[1] states that through the use of Cosme's paste (acid. arsen., 1; hydrarg. sulph. rub., 5; ungt. aq. ros., 40) and Esmarch's paste (acid. arsen., 1; morphia sulph., 1; hydrarg. chlor. mit., 8; pulv. acacia, 48) the inflammatory action is insufficient, and the danger of absorption through the low degree of concentration great. Strong enough is Bougard's paste (hydrarg. chlor. cor., 1; hydrarg. sulph. rub., 10; ammon. chlor., 10; farin. trit., 120; amyli, 120; zinci chlorid. cryst., 120), applied for 24 hours immediately after curetting. The arsenical solution of Czerny and Trunecek (acid. arsen., 1; alcohol, 75; aq., 75) is slow and tedious. Hue recommends the hypodermic injection of arsenic into the growth every 3 days, as follows: Acid. arsen., 1; cocain hydrochlor., 5; aq., 500; but the results are usually relative. The arsenic applications, especially Marsden's paste (acid. arsen., 50; pulv. acac., 50), are the best, used after curetting.

**Treatment of Skin-cancer without Operation.**—In the section on Cutaneous Medicine of the American Medical Association, H. W. Stelwagon[2] spoke of the superiority of an arsenical paste in preference to any other local application. This drug exerts an elective influence upon diseased tissue, sparing in large part the normal structure. The scarring after the use of arsenic is therefore often inconsiderable. Cocain added to the water used in making up the paste lessens the pain, which in some cases is marked. Stelwagon notes that orthoform, if added to the paste to relieve the pain, might interfere with the action of the arsenic. In discussion, Gottheil also favored arsenic. Other caustics do not spare the healthy tissue, and therefore increase the deformity by greater scarring, while arsenic in from 33% to 50%, applied up to 36 hours, will not injure the unaffected tissue. The intense inflammation induced will break down the neoplastic cells, but the older normal cells survive. Curetting is preferred by Sherwell, and this is followed by a 5 minutes' application of the acid nitrate of mercury. While painful, it is not long continued so, and with arsenic it is claimed that the distress increases. When the penis or the external ear is affected, then he advises the knife. Corlett for 9 years has used electrolysis, after which he claims never to have seen a recurrence, and for small growths a single sitting is reported as completely destructive to the lesion. C. Allen advises arsenic, and especially in cases with deep lesions. Orthoform he finds good, and employs this formula: Arsenic, 1 or 2 parts; orthoform, 1 part; rub together and add water sufficient to make a paste. W. Brayton would curet, and, after applying pure carbolic acid as an anesthetic, would follow by the acid nitrate of mercury.

**Blastomycetic Dermatitis and Epithelioma.**—W. E. Coates[3] reports a case clinically and histologically resembling epithelioma, in a man 37 years old. An attack of rheumatism came on 3 months before he applied for treatment for the lip, and during convalescence the growths appeared on the eyelid and lip. The growth had been removed from the outer margin of right lower eyelid one week previously. The growth on the lip was diagnosed as epithelioma and removed, the wound healing promptly. Eighteen months later the patient again came under observation. He stated that after having the growth removed from the lip, about 8 small nodules made their appearance on the lower extremities, mostly on the thighs. The largest was about ⅜ of an inch in diameter. They were hard and shot-like, blood-red in color, contained no pus, and at the end of 4 or 5 weeks became scaly and could be brushed off. The glands back of the angle of the jaw on the side became enlarged about 2 weeks before the original growths appeared, but disappeared after a month. A microscopic examination of the growth removed from the lip showed it to be an epithelioma. A few giant cells were observed and led to the suspicion of tuberculosis. A further examination, however, revealed the presence of numerous blastomycetes. Many additional slides of the growth were prepared, and the blastomycetes could be plainly demonstrated.

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[1] Frauenarzt, No. 1, 1900.

[2] Phila. Med. Jour., June 9, 1900.

[3] Medicine, Feb., 1900.

**Treatment of Cutaneous Cancer.**—Moreau[1] reviews the usual methods, involving ointment, pastes, heated and iced water, hypodermic injections of liq. potassii arsenitis, iodid of arsenic, etc., and claims that they fail to benefit. He points out that curetting or cauterization offers the only possible value in sarcoma and in cancer of the skin. The former he views as surgical in procedure, and thinks it less desirable. The latter is good if the galvanocautery or, better, the thermocautery of Paquelin is chosen. The method used is that recommended by Besnier: interstitial and extended ignipuncture, with care to penetrate deeply into the places where is found the zone of extension of the lesion. A subsequent application of ammonol completes the treatment. Forty-two cases are reported as cured. The sittings were two, at an interval of 15 days. For 3 days after each there were applied compresses of ammonol 4%; then for 4 successive days the strength was increased to 10%. In 2 weeks after the second radical ignipuncture the scar was complete.

**Rhinoscleroma.**—S. Rona[2] states that this rare disease is encountered most frequently in Russia, Austria, Central America, and Hungary. He collected the histories of 21 cases in Hungary. Attention is called to the observation that the regional lymphatic glands may be affected by the process. In Rona's case both submaxillary bones were enlarged and hard. A microscopic examination of the extirpated glands showed a subacute inflammatory process, and from the lymph of the glands pure cultures of the rhinoscleroma bacillus were obtained.

**Mycosis Fungoides.**—W. Dubrenilh[3] reports a case of this disease, occurring in a man aged 44, in which there was no prefungoid stage, the tumors being the first manifestations of the disease. Several months after the tumors appeared urticarial and erythematous lesions occurred.

[1] Ann. de la Policlin. de Toulouse, Mar., 1900.

[2] Arch. f. Dermat. u. Syph., 49, 1899, p. 265.

[3] Jour. des Mal. Cutan. et de Syph., Oct, 1899.

Galloway and Macleod[1] describe 3 cases and discuss the pathologic anatomy as follows: In the lesions of the prefungoid stage there exists a connective-tissue cell proliferation around the blood-vessels of the subpapillary and papillary layers, the hair follicles, sebaceous glands, coil-ducts and occasionally the coil glands, and forming foci independent of these structures situated among the connective-tissue bundles. In the epidermis active mitosis of the prickle cells and downgrowth of the interepithelial process is noted; also nests of corium tissue in the mucous layer, and interepithelial edema going on to the formation of reticular spaces. In the tumor stage the cell proliferation increases, and the cells show a marked tendency to break down, as evidenced by the crenation, irregularity, and fragmentation of the cells. The granuloma encroaches on the downgrowing epithelium, flattens it out, spreads up to the surface, and is covered only by a layer of the stratum corneum. The diseases with which mycosis fungoides have been confused histologically are the granulomas of tubercle and syphilis, the sarcomas, and the leukemic and pseudoleukemic growths of the skin. It resembles the syphilitic formation in many particulars. In contradistinction to tuberculosis of the skin, giant cells with central caseous degeneration are not met in mycosis fungoides. In the skin lesions of leukemia there is great edema, affecting the vessels of the cutis, and active diapedesis from them, and an infiltration of the neighboring cutis with leukocytes; it is purely a leukocytic infiltration, without marked fixed-cell proliferation, mitosis, or imperfect giant-cell formation such as occurs in mycosis fungoides. Pseudoleukæmia cutis is more like a syphilitic granuloma than mycosis fungoides. The bacteriologic examination proved negative.

A. P. Biddle[2] gives a report of a case, which, because of its rarity and the full account of the course of the disease, is of value. The dubious character of the early symptoms is dwelt upon and the ulceration with free oozing of offensive matter noted. No portion of the skin surface or mucous membrane is free from attack, but postmortem examination (Hyde) has not demonstrated any visceral implication. The greater number of cases occur after the age of 35. The pathogenesis is taken up and the differing opinions presented, and this confirms the remark of Unna, who states that the usual multiformity is characteristic. Possibly this disease is a variety of sarcoma, in spite of its relation to age and its prefungoid stage. The treatment varies. Iodol, boric acid, aristol, iodoform, and pyrogallic acid ointment may be used. The knife affords a quick and sure method, though recurrence is not infrequent. In this particular case, after a little less than a year no further symptoms developed.

**Observations Concerning Leukemic Lesions of the Skin.**—Oertel[3] reports a case of leukæmia cutis, and draws attention to the rarity of the condition. The first case reported was that of Biesiadecki, published in 1876. The cases of leukemia of the skin which have been published up to the present may be divided into 3 classes: The first class is represented by cases in which there are circumscribed, multiple, pin-head to hazelnut-sized, rapidly growing, pale or faintly red to brownish-colored tumors, irregularly distributed over the body, with little tendency to retrograde metamorphosis or ulceration. The second class is characterized by a few solitary, brown, markedly elevated, lobulated, firm, slowly growing, and persistent nodules. According to Neuberger, the condition consists of a lymphoid-celled infiltration into the cutis, especially around the follicles, by which the deeper layers of the epidermis are compressed, although they are still separated from the infiltration in the lower part of the cutis by a small, narrow, noninfiltrated zone of connective tissue. The third class includes those cases in which the lesions are more diffuse. They show, on the one hand, a diffuse, moist, eczematous appearance, especially of the head, extremities, and chest, and, on the other hand, tumors the size of a pea to a pigeon's egg, which ulcerate spontaneously, leaving a large, flat, red ulcer. An eczematous condition of the skin is present. The most definite statements yet recorded concerning the structure of leukemic cutaneous nodules are to be found in Nékám's recent discussion of the subject. Nékám is fully convinced that a number of the cases reported as examples of leukæmia cutis, and particularly the diffuse lesions falling under the third group, can not be regarded as such. He accepts as undoubted examples only the cases of Biesiadecki, Hochsinger and Schiff, Neuberger, and his own, in all of which there were definite nodules the size of a pea to a hazelnut without a diffuse appearance. Nékám states that we can therefore accept as such only those which fulfil the following conditions: (1) They must occur during the course of true leukemia; (2) their origin must be exclusively a diapedesis of cells from the blood, local proliferation of cells being absent; (3) a part of the cells, especially the red corpuscles, are returned to the blood by way of the lymph-channels; another part, especially the leukocytes, are carried off through the epidermis, or otherwise discharged; (4) the chief bulk of the cells composing the nodules undergo no metamorphosis. Oertel's case was a man, aged about 40, who had been ill for 2 years, thought to be suffering with malaria, owing to the markedly enlarged spleen. Some time before the patient's death, "there appeared on the skin small nodules, irregularly distributed over the body." Their appearance at once led to a diagnosis of multiple sarcoma of the skin. A blood examination was ordered, and a slide showed an enormous increase in the white corpuscles, with numerous myelocytes and eosinophiles, the diagnosis now being leukemia. The patient died. The lower part of the cutis was the seat of a dense and diffuse so-called small-celled infiltration, the cells presenting the characteristics of lymphocytes, which were of two kinds: (1) Small cells with narrow, faintly staining cytoplasm and deeply staining nucleus, and (2) larger cells with feebly staining cytoplasm and a large, sometimes indented, somewhat irregularly staining nucleus; also (3) polymorphonuclear leukocytes and (4) eosinophilic leukocytes. There were but few red blood-corpuscles and only an occasional plasma cell.

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[1] Brit. Jour. of Dermat., May and June, 1900.

[2] Physician and Surgeon, Jan., 1900.

[3] Jour. Exp. Med., Sept.-Nov., 1899, vol. IV, p. 369.

No mitoses were observed, and in this regard Oertel's observations agree with Nékám's. Oertel states that his study of the nodules in this case goes to support the view of those who regard secondary leukemic nodules as essentially composed of cells derived from the blood rather than from the local proliferation of cells.

**Leukemic Tumors of the Skin.**—Karl Kreibich,[1] in a woman of 58, noticed a tumor-like swelling in the region of the eyebrows, sharply separated in its upper part by the middle fold of the forehead, and passing into a diffuse swelling of the nose in its lower part. Both areas were occupied by ovoid growths, and upon the skin a tumor of the size of a pigeon's egg existed. The tumors were movable with the skin and were of a brownish color. The skin covering the tumors was shiny and smooth, interwoven with large vessels, and gave a sensation of velvet to the touch. The tumors did not ulcerate, but side by side with them a diffuse form of leukemic affection of the skin existed, presenting the clinical features of an infiltrated, moist eczema. The glands of the neck reached the size of walnuts, and the cubital glands were also enlarged. The liver and the spleen were enlarged and on the abdomen tumors of the size of a fist existed. The proportion between the white and red blood-corpuscles varied between 1 : 28 and 1 : 20. The small mononuclear white blood-corpuscles were especially increased in number. Large mononuclear, multinuclear, polymorphonuclear, transitory cells, and eosinophiles were present. The process began between the corium and the fatty tissue. The cells composing the tumor consisted of mononuclear cells, with a small amount of protoplasm, the bulk of the tumor containing mostly collagenous fibers. The blood-vessels and lymph-vessels were filled up with mononuclear cells. The macroscopically unchanged skin, when examined microscopically, revealed changes in its deep vessels and around small vessels, which were like the changes in well-developed tumors. The lumina of the vessels contained numerous leukocytes and heaps of mononuclear and megalonuclear cell elements.

**The Alterations of the Skin Occurring in Lymphatic Leukemia and in Pseudoleukemia.**—Pinkus [2] concludes a paper upon this subject as follows: The tumor-like localizations occurring in the skin in lymphatic leukemia can not be distinguished from those occurring in pseudoleukemia; they are clinically and histologically identical. The cases of so-called mycosic erythrodermia stand in close relation to the leukemic skin localizations, which are not to be reckoned as belonging to mycosis fungoides, but as belonging to the lymphodermia perniciosa of Kaposi. These form a group which is characterized by erythrodermia (an eczema-like condition with redness, swelling, itching), the occasional occurrence of marked thickening of the skin, and in some cases actual tumor formation, and the coexistence of lymphocythemia. The diagnosis of such cases can be definitely made only after the appearance of the skin and lymph-gland tumors. Histologically the leukemic skin tumors consist of a collection of lymphocytes in the corium and subcutaneous tissue, which, at the site of the tumors, arises from previously normally present lymphatic tissue, and not from lymphocytes supplied by the blood-vessels. It is, in a certain sense, a lymphatic granulation tumor.

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[1] Arch. f. Dermat. u. Syph., vol. XLVII. pp. 185–194, 1899.

[2] Arch. f. Dermat. u. Syph., Bd. I, Heft 2, 1899.

**"Fungus Foot."**—J. T. Arwine and D. S. Lamb[1] report the fifth case of this disease in America. The patient was 45 years of age, born of Mexican parents, and lived in southwestern Texas. The disease began about 1887, with severe pain in the left foot, lasting 36 hours, after which a red spot appeared on the foot. There were chills and fever. The pain continued throughout the course of the disease. The local physicians diagnosed elephantiasis and advised amputation. Twelve years after the onset of the trouble the leg was heavy, but there was no pain. An amputation was made, and the arteries were found to be considerably enlarged. The amputated portion was examined at the Army Medical Museum, and was found to be a typical case of mycetoma, or Madura foot. The foot and ankle were much enlarged and showed the characteristic "buttons" of fungus foot. They varied in size, were closely set, and were numerous on the dorsum of the foot. The subcutaneous tissues showed many interstices, filled with an oily, white, pasty matter mixed with brownish bodies measuring from 0.5 mm. to 2 mm. in diameter. The author says that it is possible that many of the cases which are diagnosed elephantiasis are really examples of this disease. The two American cases in possession of the Army Medical Museum came from Texas. In India the disease is found almost exclusively among the peasantry, who go barefoot on the soft, bare ground.

## THERAPEUTICS.

**Strong Heat as a Palliative of Pruritus.**—E. Andrews[2] gives quotations from many advising hot water, or other forms of great heat, as a means of cure or of relief for itching. He states that the farmers in the north, who suffer much from chilblains, expose these lesions to close radiation of heat from live coals, and cure follows after 4 or 5 such daily treatments. Napoleon I, of France, used hot baths as a means of relieving the pruritus of eczema, he being one of the first on record to use this method. Among modern authors recommending this are F. Treves, of London, Matthews, of Louisville, and Kelsey and L. D. Bulkley,[3] of New York. Pruritus of scrotum is relieved by immersion in hot water. For pruritus ani, among other methods of treatment is that of dilation, a plug being worn all night. A case of psoriasis, which was treated ineffectually by arsenic and other drugs, was reported cured by using small streams of very hot water after the scales were scraped off.

**Use of Chrysarobin.**—M. Hodara[4] gives the following formula, of value in pityriasis seborrhœica (the symptoms of which are yellow, dry patches, raised and desquamating, disseminated over face, upon ears, etc.), of value also in seborrheic eczema: Yellow vaselin, 50 parts; chrysarobin, 0.01 to 0.05 parts; ichthyol, 0.08 to 0.2 parts. In place of the 50 parts of yellow vaselin, when an ointment is not desired, a lotion is made by substituting alcohol 100 parts, and instead of the ichthyol, ol. ricin. 0.5 to 2.0 parts may be used. This prescription is to be used once a day. In cases of psoriasis he suggests the following: Chloroform and glycerin, āā 25 parts; chrysarobin, 2.5 parts; ichthyol, 2.5 parts or less; and ac. salicylic., 2.5 parts. This is used every third day, and between he recommends the application of ol. olivæ, or of ung. aq. rosæ, this latter alone or with the addition of 1% ichthyol. By this method facial lesions are cured, and without the irritative effects common to chrysarobin.

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[1] Am. Jour. Med. Sci., Oct., 1899.

[2] Clin. Rev., May, 1900.

[3] Jour. Am. Med. Assoc., April 16, 1898.

[4] Jour. de Méd. de Paris, Dec. 17, 1899.

**Hydrogen Dioxid in Skin Diseases.**—L. D. Bulkley [1] speaks of the great value of this remedy in cutaneous affections, its action being parasiticidal and antiseptic; the value is considerable for suppurating surfaces and within cavities. Among diseases in which it may be successful ulcer of leg, epithelioma (when superficial), lupus after operation, and chloasma are cited. Its bleaching properties render it useful in hiding superfluous black hair upon the face of women. It retards the growth of hair, and for this can be used in hirsuties. Electrolysis is of value in connection with its use. When applied in chloasma, it is to be used freely, allowing it to soak in, as in the treatment of ulcers. If desquamation occur, this is relieved by lanolin and rose ointment or by borax and glycerin lotion.

**An Ethereal Solution of Soap.**—Herbert Skinner,[2] pharmacist to the Great Northern Central Hospital, gives the following for an ethereal solution of soap, which may be made extempore: ℞. Oleic acid, methylated alcohol, of each, ½ ounce; strong liquor ammoniæ, q. s.; methylated ether to 2 ounces. The first two are mixed and the ammonia is added in order to neutralize the acid; care should be taken not to add an excess. A little heat is evolved, but nothing to speak of. Then add the ether, which may be increased to 3 ounces if a weaker solution is required. Petroleum spirit or benzine may replace the ether if desired. It is excellent for cleansing ointment-laden surfaces, being far superior to any ordinary soap. It is better adapted for a physician's use in examination than for a patient's. Caustic tar preparations, such as lysol and creolin, are not excluded, but naturally should be sparingly used, as an ethereal solution is very penetrating. It can be rendered antiseptic, not with mercuric chlorid, but with the biniodid freshly prepared—that is, mercuric chlorid dissolved in a strong solution of potassium iodid.

**A Cream of Zinc Carbonate.**—Herbert Skinner [3] says that zinc carbonate is preferable to commercial calamin, which is often impure, and far from desirable as an application to an inflamed surface. Zinc carbonate compounded as follows makes a useful so-called cream: ℞. Glycerin amyli, adeps lanæ hydros., zinci carb., glycerin, of each,

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[1] Jour. Am. Med. Assoc., Dec. 23, 1899.

[2] Brit. Jour. of Dermat., May, 1900.

[3] Brit. Jour. of Dermat., May, 1900.

℥ss. The first two are mixed together, then the second two, and finally both together. The quantities may be increased or decreased according to the consistence desired.

**Gelatin Applications.**—Herbert Skinner[1] experimented with gelatin and agar-agar, but the latter was not found desirable for the purpose. Gelatin is more tractable, but any formula given will depend on the quality of the gelatin used, no two samples seeming alike, while half an hour's heating will make two samples from the same lot behave differently.

**X-rays in the Treatment of Skin Diseases.**—W. A. Pusey[2] states that the conditions to be avoided are: (*a*) Too great strength of primary current. A current of more than 1½ amperes and 12 volts' strength should never be used. The currents of high amperage used in skiagraphy should never be employed. (*b*) An inductor of more than 30 cm. spark length should never be used. (*c*) Too long and too frequent exposures should not be used. (*d*) The distance between the skin and the tube should be considerable. In the beginning, the sittings should not be longer than 5 minutes, and the distance of the tube not less than 15 cm.

**X-rays in the Treatment of Skin Diseases and for the Removal of Hair.**—William Allen Pusey[3] states that the effects of the rays upon the skin and subcutaneous tissues are all inflammatory in character, ranging from slight erythema to violent inflammation ending in necrosis. The two actions of the x-rays on tissues which offer the most promising prospects therapeutically are: (1) its power of causing the falling out of the hair; and (2) its power of causing inflammatory reaction and thus influencing the nutrition of connective tissue. The practical problem is the application of the rays in such a manner that undesirable results may be avoided. One should not use a current of more than 1½ amperes and 12 volts' strength, this current, as is well known, being much weaker than that employed for skiagraphy. An inductor of more than 30 cm. spark length should not be used, nor should the exposures to the rays be too long or too frequent. At the beginning the sittings should not be longer than 5 minutes, and the distance of the tube not more than 15 cm. Freund, who has done considerable experimentation upon the skin, recommends for the attainment of the best results the use of a mechanical interrupter run at the rate of from 800 to 1000 interruptions a minute. Suitable lead masks for protecting surfaces which are contiguous to the areas to be treated should be used. The evidences that the exposures have been carried far enough are the appearance of erythema or pigmentation, the blanching of the hair, and the loosening of the hair. The application of the x-rays has been chiefly in 4 classes of affections: In hypertrichosis—for the removal of hair; in diseases of the hair and of the hair follicles; in inflammatory diseases, like chronic eczema; and in certain specific diseases, like lupus.

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[1] Brit. Jour. of Dermat., May, 1900.

[2] Jour. of Cutan. and Genito-urin. Dis., Aug., 1900.

[3] Jour. of Cutan. and Genito-urin. Dis., July, 1900.

The author believes that the agent is a valuable one for the removal of hair, especially in cases when the growth is diffuse and profuse. In lupus the rays seem to have a selective action, and employed properly constitute a remedy of value.

**Cooling Paste.**—Unna[1] recommends the following in place of the well-known zinc oxid paste in the early treatment of painful inflammatory conditions of the skin, and in critical cases of unknown nature in which relief is needed immediately without risk of modifying the character of the skin disease present: ℞. Ol. lini, aq. calcis, āā 20; zinci oxidi, cretæ præp., āā 30. The combination is a good type of a wash containing a powder and a fat.

**The Anesthetic Properties of Nirvanin.**—C. A. Elsberg[2] considers nirvanin preferable to eucain or cocain in the production of local anesthesia by the infiltration method. The drug is one of the products obtained in the synthetic formation of orthoform. It is a white salt, without odor, soluble in water. The anesthesia is confined to the infiltrated area. The drug possesses no irritating effect upon the tissues. It has distinctly antiseptic properties, and is stable. It is said to be 10 times less poisonous than cocain and 3 times less so than eucain.

**A Painless Application of Arsenical Paste.**—W. P. Nicholson[3] states that while morphin and cocain have been only of slight assistance in reducing the pain caused by the topical escharotic use of arsenic, orthoform obviates the pain in a marked degree. The formula recommended consists of equal parts of arsenous acid, powdered acacia, and orthoform, made into a paste and applied to the surface to be destroyed. The anesthetic effects last several hours.

**The Result of the Treatment of Skin Diseases with Tuberculin R.**—Napp and Grouven[4] report extensive trials of the new tuberculin. The tuberculin was at first injected with normal salt solution; later with water containing 20% of glycerin. Most of the lesions were covered with moist antiseptic dressings at the same time that the tuberculin treatment was carried out. The fever was of the well-known irregular type. Twice it happened without apparent cause that two strong young men presented alarming symptoms of cyanosis, dyspnea, collapse, etc. All the patients lost in weight. Albuminuria was insignificant. One patient developed icterus and cutaneous hemorrhages. The first dose used was as recommended by Koch, but each subsequent dose was about $\frac{1}{5000}$ of a milligram greater. Local reaction was only once observed. A number of patients had recurrences after improvement. The authors conclude that tuberculin R, even if it does not produce a permanent cure, still has a markedly favorable influence in that it attacks the tuberculous process at the same time that it produces, if used with care, no serious disturbance of the general condition. This report is as favorable as any which has been given concerning its use in dermatologic cases.

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[1] Monatsh. f. prakt. Dermat., 1900, Bd. XXX, Heft 1.

[2] N. Y. Med. Jour., Jan., 1900, p. 47.

[3] Atlanta Jour.-Rec. of Med., vol. I, p. 738.

[4] Arch. f. Dermat. u. Syph., Bd. XLVI, Heft 3.

**Thiosinamin in Dermatology.**—J. T. Bowen[1] gives Unna's experiments with thiosinamin soaps and plasters. Hebra had observed that this substance injected subcutaneously had a marked action on the lupous process, particularly the scar tissue. The latter was rendered pliable, so that joints which had been immovable by contraction of surrounding scars recovered complete motion. The substance being insoluble in water, and injections of alcohol solution being painful, Unna used it in the form of a soap, in 5%, 10%, and 20% strength, and spread on plaster muslin, in the treatment of fibrous tumors of various kinds, keloids, leprous and syphilitic lesions, and scarring from smallpox. He found them much more effective in the treatment of these conditions than his former treatment, consisting of massage combined with mercurial plaster, salicylic acid plaster muslins, etc., had been. With these plasters no irritation or pain was experienced, as had been the case with injections. In the treatment of smallpox scars a mask of thiosinamin plaster was worn at night. The parts of the body free from hair and protected by clothing were benefited most by a plaster worn constantly. The soap is more effective on the face, hands, and scalp, it being allowed to dry on. Severe cases may be treated by the plaster at night and the soap by day. This treatment may be alternated with mercurial plasters and massage. Unna believes that thiosinamin is a distinct advance in the therapeutic treatment of fibrous cicatricial deformities, which are regarded by most practitioners as practically hopeless.

**Treatment of Pruritus Ani.**—L. H. Adler, Jr.,[2] gives a method of treatment for the uncomplicated form of this disease, using a daily injection of from 1 to 2½ drams of the following: ℞. Ext. hamamelis fl., f℥j; ext. ergot. fl., ext. hydrast. fl., tr. benzoini comp., āā fʒij; ol. oliv. (carbolat. 5%), f℥ij. Shake well before using. Do not allow the patient to give way to the feeling for an evacuation of the bowels which follows this injection by leaving the examination table; this feeling is due to the alcohol in the solution, and soon passes away. Also inunctions of ung. hydrarg. nitratis are employed. Applications of very hot water are used at night, if the pruritus occurs, and then a solution of black wash or of calomel ointment is given locally as a sedative.

## SYPHILIS.

**Contagion from Syphilis of 13 Years' Duration.**—Herscher,[3] at a meeting of the Société de Dermatologie et de Syphiligraphie, exhibited a woman who had just been infected by her husband, who had contracted syphilis 13 years previously. The exhibitor had himself seen ulcerations upon the penis of the man which were in full activity at the time infection occurred. The woman had a chancre in the region of the genitalia. The case was apparently one of contagion from a syphilis of 13 years' duration.

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[1] Boston M. and S. Jour., Feb. 22, 1900.

[2] Phila. Med. Jour., May, 1900.

[3] Gaz. hebdom. de méd. et de chir., Feb. 8, 1900.

**Absence of Teeth as a Sign of Hereditary Syphilis.**—Fournier,[1] in exhibiting a young boy who was a typical example of hereditary syphilis, being puny, with a leaden complexion and characteristic tibial deformities, called attention to the absence of two superior lateral incisors. A study of the observations upon hereditary syphilis shows that in a great number of cases 1, 2, 3 and sometimes 4 teeth are absent. It is, therefore, necessary to look not only for anomalies of form, direction, and structure, but to take into account also the absence of certain teeth as stigmata of hereditary syphilis.

**Dental Malformations and Hereditary Syphilis.**—Galippe,[2] at a meeting of the Société de Dermatologie et de Syphiligraphie, read a note upon the importance of dental malformations as signs of hereditary syphilis. He does not believe that dental malformations characteristic of hereditary syphilis exist. Heredity is the great factor in these malformations. Hutchinson's teeth are a great rarity, if they exist. The peculiar "screw-driver" teeth do not differ notably from certain normal teeth, these structures differing greatly according to race and individual. All dental dystrophies which have been regarded as belonging to hereditary syphilis may be encountered in all degenerative conditions, whatever may be their cause.

**The Contagiousness of Inherited Syphilis.**—Kolipinski [3] reports 2 instances in which a syphilitic infant infected previously healthy individuals. In the first case a young woman, a year after having been infected by her husband, gave birth to a syphilitic infant, which died 2 months after its birth from syphilitic pemphigus. During its illness this infant was taken care of by the grandmother, a widow of over 60, who frequently inserted her finger in its mouth, which was sore, for the purpose of removing mucus and quieting it. Shortly afterward the grandmother presented an initial lesion on the index-finger of the right hand. Subsequently she presented the ordinary symptoms of secondary syphilis, and, in addition, suffered from deafness and two attacks of iritis. In the second case a young woman, who had been treated for a chancre of the left labium majus followed by the usual secondary symptoms, gave birth to a syphilitic infant. The mother dying from puerperal septicemia, the grandmother took care of the child. Three months later she had a chancre on the right ring-finger, which was followed in due time by secondary symptoms. The infant died at 6 months from syphilitic meningitis.

**Syphilis Rebellious to Specific Treatment.**—Fournier,[4] at a séance of the Société de Dermatologie et de Syphiligraphie, presented a woman who had contracted syphilis 2 years previously from her husband. During this period she had suffered from syphilitic symptoms of a severe character, notwithstanding the administration of mercury and the iodids. He also referred to a like obstinate case in a man. In both, mercury had been given by the mouth, by inunction, and by intramuscular injections. The woman had had 40 injections of calomel and the man 120 without any curative effect. The mother of the woman had died of phthisis and her father from absinthe-drinking, which might in some degree explain the lack of success, although similarly obstinate cases were met in which there was no such history. A certain amount of benefit had been obtained from serum injections.

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[1] Gaz. hebdom. de méd. et de chir., Jan. 18, 1900.
[2] Gaz. hebdom. de méd. et de chir., Jan. 18, 1900.
[3] Maryland Med. Jour., Nov. 25, 1899.
[4] Ann. de dermat. et de syph., July, 1899.

**A New Method of Treating Syphilis by Inhalation.**—Kutner,[1] believing that the beneficial effects following mercurial inunctions in syphilis are largely due to the inhalation of mercurial vapor disengaged by the friction, rather than to the absorption of the mercury by the skin, has devised the following method of treatment: Mercurial ointment is thoroughly rubbed up in a closed box and the mercurial vapor thus produced is inhaled by the patient through a mask and a tube leading from the box. The apparatus is so arranged that the temperature of the interior of the box may be raised if this is desired. The advantages of this method of inhalation are: Utilization of all the vapor disengaged; exactness of dosage; shortness of the period during which the mercury is inhaled, and, in consequence, less inclination to stomatitis; its convenience as compared with other methods. After the use of such inhalations the author was able to demonstrate the presence of mercury in the urine. The therapeutic results were quite satisfactory.

**Postconceptional Syphilis.**—Fabre and Patel[2] at a meeting of the Lyons Society of Medical Sciences presented photographs representing sections of placentas from cases of postconceptional syphilis, infection having occurred in the third, fourth, and seventh months of pregnancy. Periarteritis and endophlebitis were constantly present on the fetal surface of the placenta. The cells in the interior of the villi were less numerous and their nuclei less deeply stained than normally. The epithelium on the external surface of the villi was markedly affected, being destroyed or proliferating. On the maternal side the decidua was hypertrophied. Its line of junction with the intervillous tissues formed cones which penetrated the interior of the chorion. The cells of the decidua were mostly abnormal, and were separated by a layer of fibrin or agglutinated. The membranes were likewise altered; there was an amniositis, the cells being irregularly nucleated and the fibers of the chorion separated.

**The Recrudescence of Syphilis Due to Influenza.**—Haward[3] thinks influenza comparable to malaria in its ability to bring out late symptoms of syphilis and to make manifest any latent syphilitic taint. He reports the case of a woman of 42 who had contracted syphilis from her husband 18 years previously, there being a clear history of an initial lesion upon the vulva followed by well-marked secondary symptoms lasting a year and a half. During the following year she was free from symptoms, being continuously under treatment; but in the succeeding year she suffered from ulceration of the throat and skin. She then remained well until an attack of influenza, but in this attack rupial bullæ appeared over the body, which developed into numerous and extensive ulcers. The hair fell out and ulceration of the soft palate occurred, causing extensive perforation. A periosteal node formed over the back of the radius, and ulcers appeared between the toes. The author has observed other cases in which there seemed to be a special tendency to ulceration. This tendency to necrosis is an evidence of lowered vitality, and should be taken into account in treatment. In such cases the calomel vapor bath will be found very useful. The recent occurrence of influenza is no bar to the administration of mercury, which will always be found to be of the greatest benefit. Good results have likewise been obtained from the simultaneous administration of a fresh decoction of sarsaparilla.

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[1] Berl. klin. Woch., Jan. 8, 1900.

[2] Lyon méd., July 16, 1899.

[3] Lancet, July 1, 1899; N. Y. Med. Jour., Aug. 5, 1899.

**Luetic Struma (Goiter).**—Wermann [1] reports the following case as showing that the thyroid gland, under the influence of syphilis, may present a clinical picture which agrees perfectly with that seen in the ordinary form of goiter: A young man of 24, who had had various manifestations of syphilis,—such as roseola, sore throat, eruptions in the mouth, double orchitis, and gumma of the palate with subsequent perforation, all of which yielded to mercurial treatment,—in the sixth year of the disease presented somewhat suddenly a swelling of the thyroid gland, which increased in size quite rapidly. The gland was moderately enlarged but painless, the middle lobe being enlarged as well as both lateral ones. There were no symptoms of Basedow's disease. Increasing doses of potassium iodid and the external use of iodinvasogen were without effect. Mercurial inunctions were then tried, and after the sixth inunction there was an appreciable decrease in the swelling. After 14 days the gland had much diminished in size. Upon the suspension of the treatment, however, it again began to enlarge, but with the resumption of the inunctions a diminution of the swelling once more occurred. The author calls attention to the possibility that syphilitic disease of the thyroid may lead to Basedow's disease or to myxedema if not treated early and properly.

**Syphilitic Edema.**—Fusier [2] describes as syphilitic edema the serous infiltration of the cellular tissues which occurs in syphilitics who present no cardiac, renal, or hepatic disease, and which rapidly improves under antisyphilitic treatment. The serous infiltration may appear at an early or a late period, and may be generalized or local. The generalized form may appear rapidly, when it is associated with effusions in the serous membranes, or it may develop in a chronic manner. The pathogenesis of this form of edema is complex; but the generalized form may usually be connected with a dyscrasia, while the local variety is due to a lymphatic cause, preferably of mechanical origin.

**Early Syphilitic Nephritis.**—Allaria [3] asserts that a nephritis due to the syphilitic virus may occur in the secondary stage of syphilis.

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[1] Berl. klin. Woch., Feb. 5, 1900.

[2] Gaz. hebdom. de méd. et de chir., Oct. 5, 1899.

[3] Clin. Mod., An. v, N. 24; Brit. Med. Jour., Dec. 30, 1899.

This form of nephritis is characterized clinically by the presence of anasarca, severe albuminuria, and recovery under specific treatment. It is partly parenchymatous, such as occurs in other infectious diseases, and partly interstitial, the latter being due to the specific virus. Tertiary syphilitic nephritis is a more chronic affection in the majority of cases, and the albuminuria is less marked.

**Precocious Hereditary Syphilis.**—Rabek [1] in 300 cases of hereditary syphilis has found that the lesions are most frequently seated upon the skin (macules 204 times, papules 41 times, with a combination of both sorts of lesions in several cases, pemphigus 3 times, and cutaneous gummas 9 times). The skin is pale, bister, as if colored by *café noir;* in the light, especially upon the soles of the feet, looking as if varnished. Upon the mucous membranes the author has noticed discrete or confluent plaques, gummas, flat condylomas of the buccal cavity and of the pharynx, and in most cases coryza and ozena. Much more rarely he has met with muscular and osseous lesions; and, in the last place, affections of the nervous centers and of the viscera, considerable swellings of the liver and of the spleen in syphilitic infants not always being of specific origin. Most of the observations were in infants at least three months old, and in almost all of them the symptoms were sufficiently plain to make the diagnosis clear. One occasionally sees precocious hereditary syphilis recur at the age of from 1 to 3 years, most frequently under the form of flat condylomas or gummas. The most active form of treatment is sublimate baths; and, next, frictions with Neapolitan ointment, and calomel internally. Artificial alimentation almost never succeeds; the infant should be nursed by its mother or by a syphilitic nurse.

**Syphilitic Secondary and Gummatous Epididymitis.**—Melle,[2] in reporting 8 cases of syphilitic epididymitis, concludes as follows concerning this affection: The epididymis may be primarily affected, without accompanying disease of the testicle, in the secondary as well as tertiary stage of syphilis. This primary syphilitic epididymitis has some characteristic features by which it may be recognized. Owing to its indolence and its insidious course it may not be recognized until it has existed for some time. It usually develops in middle age, after 25. In recent syphilis it is always bilateral; in the gummatous form it is frequently, but not always, unilateral. In secondary syphilis the affection is always localized in the head of the organ, but gummatous syphilis affects the whole epididymis. The disease of the head in secondary syphilis, in the course of time, is associated with disease of the cauda of one or both sides; in tertiary syphilis this is not constant. Gummatous epididymitis is in one-half the cases accompanied by a unilateral gummatous deferentitis. Primary syphilitic epididymitis is less frequent in the secondary than in the tertiary stage, but in neither stage can it be considered a rare affection. It may be the only manifestation of syphilis, but it usually occurs in connection with other symptoms. Its occurrence is no indication of severe syphilis; and recovery takes place under antisyphilitic treatment. In affections of the epididymis of doubtful nature it is always proper to try antisyphilitic treatment.

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[1] Kron. Lek. et Przeglad Chir., tome IV, Zeszyt II; Jour. de Méd. de Paris, July 2, 1899.

[2] Giornale Ital. delle Mal. ven. e della pelle, 1898, XXXII.

**Syphilis of the Stomach.**—Editorially the "Journal of the American Medical Association," on March 24, 1900, expresses a doubt as to the excessive infrequency of this morbid process. Flexner [1] reports a case of gummatous infiltration, the first in English literature. Fourteen other cases have been collected altogether. Fraenkel describes a case of acquired syphilis of the stomach and intestines. Aristoff [2] claims that microscopic lesions of the stomach are frequent in hereditary syphilis, giving reports of 9 cases. Cesaris-Demel [3] gives one—acquired and of the ulcerative type. The clinical diagnosis in this had been round ulcer. This was what the necropsy disclosed, but microscopically the appearance was as is usual in specific changes of other viscera—changes in the vessels and marked gummatous infiltrations. The ulcers are usually multiple, the result of disintegration of the gummas, and also due to circulatory disturbance. Death may result from hemorrhage. Dieulafoy, Fournier, Mackay, and Einhorn report recovery from gastric ulcer occurring in syphilis, which they claim was due to the specific treatment. In syphilitic tumor of the stomach the symptoms are those of a malignant growth, resembling cancer, but curable by specific treatment. Two cases of pyloric stenosis are reported so cured, the tumor becoming not palpable.

**An Experiment in the Transmission of Syphilis to Calves.**—Ravenel,[4] having selected two calves,—one a heifer 8 months old, the other a bull 14 months old,—endeavored to inoculate them with syphilis, using scrapings from a mucous patch of the lip and from a genital sore upon a patient in the early secondary stage of syphilis. The method used was that employed in the production of vaccine virus. The selected area after being shaved was thoroughly washed with soap and water, rinsed well with sterile water, and then scraped with a sharp scalpel until bloody serum began to exude. It was next scarified, and the syphilitic material was then well rubbed in for 5 minutes. Very slight inflammatory reaction followed, and the crust which formed dropped off on the ninth day. One calf was killed 54 days after inoculation, when it was impossible to discover any evidences in the least suggestive of syphilis. The second calf was killed 138 days after inoculation, and in this animal the organs were found to be entirely normal except one lung, which contained a tuberculous nodule. The results of these experiments confirm the conclusions of other observers who have failed in their attempts to transmit syphilis to the lower animals.

**The Justus Blood Test for Syphilis.**—D. H. Jones [5] has employed this test in 53 cases, 35 of which were syphilitics and 18 were control cases. This test is based upon the statement of Justus that in untreated cases of secondary, tertiary, and congenital syphilis a single mercurial inunction causes a reduction in the hemoglobin. Of the syphilitics, 17 were cases of active syphilis not under treatment; 1 was a case of active syphilis under treatment; 2 were latent cases; 8 were cases of chancre with adenitis; and 7 were cases of chancre without adenitis. Of the 17 active, untreated cases, 13 reacted positively to the test and 4 negatively. Of the 4 negative cases, 3 had a characteristic syphilid and the fourth had a mucous patch and sore throat. The active case under treatment, as well as the two latent cases, did not respond to the test. Of the cases of chancre with adenitis, only 2 responded, while 6 were negative. Of the 7 cases of chancre without adenitis, only 1 gave a positive reaction. In all the control cases the test was negative. The author concludes that, while the test has value in doubtful cases, it is not infallible. The test also often fails in latent cases and in early chancre, and sometimes at the beginning of the secondary stage.

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[1] Am. Jour. Med. Sci., 1898.

[2] Zeit. f. Heilk., 1898, XIX, 395.

[3] Arch. per le Sci. Med., 1899, XXIII, 269.

[4] Am. Jour. Med. Sci., April, 1900.

[5] N. Y. Med. Jour., April 7, 1900.

**The Relationship of Leukoplakia to Syphilis.**—Janovsky,[1] from a study of 50 cases of leukoplakia, concludes as follows: Besides the leukoplakia, which stands in a remote or genetic relationship to syphilis, there is a form which has nothing to do with syphilis; which develops in diabetes, dyspepsias, and gastro-intestinal diseases generally, or, in many cases, independently of these affections through direct irritation of the tongue or the buccal mucous membrane by tobacco or alcohol. Gout and rheumatism play no part in its production. A genuine leukoplakia, however, which is not necessarily to be regarded as syphilitic, occurs in individuals who have had syphilis. There is also a form which develops upon a syphilitic basis, either through changes in places which previously were the seat of syphilitic processes or in situations not so affected. This form is seldom influenced by antisyphilitic treatment, and should be designated as a parasyphilitic affection. In the treatment of this malady a distinction must be made between the mild and severe cases, between the syphilitic and nonsyphilitic forms. In the mild forms tobacco should be used in great moderation, if at all; in the severe cases its use should be strictly forbidden. Highly spiced, very hot, or very cold food should be avoided. The condition of the teeth should be carefully looked after, all carious teeth being removed or filled. Strong alcoholic drinks must be avoided. Careful cleansing of the mouth is very important. For this purpose mouth-washes of sodii bicarb., 3 % or 4 %; potash chlorate or salol, ½ % or 1 %; or, in syphilitic cases, a ⅕ % solution of chromic acid, may be used. The author has also found a 1 % to 3 % solution of soda salicylate useful. In the cases in which the leukoplakia has followed directly upon a syphilitic eruption general treatment by means of inunction or injection will be effective. In mild nonsyphilitic cases lactic acid may be applied, penciled over the patches; or balsam of Peru, which in a series of cases has been found brilliantly effective by the author. In severer cases a resorcin paste is a suitable application, used twice a week. In the severest cases excision or the Paquelin cautery may be used.

[1] Wien. med. Woch., Nos. 48, 49, and 50, 1899.

**Treatment of the Local Accidents of Syphilis.**—Hallopeau[1] recommends the use of caustics in the treatment of the local affections of syphilis when it is desired to produce a profound and energetic action, acid nitrate of mercury and sublimate in powder being the agents most in use. The acid nitrate of mercury is a truly heroic remedy against syphilitic affections of the mucous membranes, but it is not employed often enough, on account of the fear of the pain which it causes. By the use of cocain, however, the pain may be rendered insignificant, and this agent should be employed regularly instead of the only moderately efficacious silver nitrate. Sublimate in powder produces a caustic effect which should be closely watched because of the dermatitis which may follow it; on this account its application should be strictly limited to the parts which it is desired to act upon. It may be used to abort chancre when the lesion is quite recent and is not accompanied by adenopathy. Permanent applications of sublimate in solution render valuable service; these may be used in the strength of 1 : 5000 and 1 : 3000, according to the sensitiveness of the patient and the amount of the reaction produced. The affected parts should be covered with compresses wet with the solution and the whole surrounded by rubber cloth. It may be applied to all syphilitic ulcerations. Being free from pain and easy of application, it is one of the surest means of producing a rapid improvement in the condition of the parts and of transforming a specific ulceration into a simple wound. Baths of sublimate act in the same manner upon the entire surface of the body. They are a convenient means of treating roseola and generalized papular syphilids. They hasten their disappearance and thus remove numerous foci of infection.

**Injections of Hayem's Artificial Serum in Malignant Syphilis.**—Augagneur,[2] in 2 cases of syphilis of malignant type which had resisted the usual treatment by mercury and potassium iodid, obtained remarkable curative effects by injections of artificial serum composed as follows: Sodium chlorid, 7 gm.; crystallized disodic phosphate, 2 gm.; water, 1000 gm. From 400 gm. to 500 gm. of this solution were injected every 5 or 6 days, while the administration of mercury and potassium iodid was continued. The effect of these injections was unquestionable. In the first case—one of malignant precocious syphilis—results were obtained which inunctions of mercury and potassium iodid were unable to produce alone. In the second case—a late malignant syphilis which developed while injections of calomel were being used—the good effects of the injections rapidly became apparent. The greater the febrile reaction, the more abundant the diuresis, the greater is the therapeutic effect.

**Treatment of Syphilis by Injections of Antisyphilitic Serum.**—Neviorovsky[3] has made some clinical observations upon patients affected with recent syphilis subjected to treatment by injections of blood-serum taken from syphilitics with tertiary lesions. As a result of these observations, as well as from examination of the blood of these patients, the author concludes that this subject should be studied, since in a certain number of cases he has noticed a quite rapid disappearance of the objective symptoms, as well as an improvement in the composition of the blood. It should be added that control experiments made with the blood of healthy individuals gave absolutely negative results.

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[1] Jour. de Méd. de Paris, Mar. 18, 1900.

[2] Ann. de dermat. et de syph., No. 5, 1899.

[3] Rev. de Thérap. Méd.-Chir.; Jour. de Méd. de Paris, Feb. 1–8, 1900.

**The Special Pathology and Reclining Rest Treatment of Syphilis.**—Friend[1] believes that all syphilitic lesions begin in the arteries, all the symptoms of the affection being explicable by acute or chronic changes in the arterial walls. This special pathology demands that reclining rest be made a routine treatment in the constitutional stage of the disease, physiologic experiment showing that when the body is at rest the volume of blood in the arteries is diminished and the arterial tension is lessened. In the ambulatory treatment of constitutional syphilis the condition of the arteries is made worse, the disease is prolonged, sequels occur, and relapses are the rule in the later stages.

**Injections of Calomel in Syphilis.**—Danlos,[2] at a meeting of the Société de Dermatologie et de Syphiligraphie, presented a patient who had contracted syphilis 3 years previously, and had had, in consequence, iritis followed by almost complete amaurosis. He had been treated by injections of biniodized oil on two different occasions without any other result than a severe stomatitis. Upon entering the service of Danlos he was treated by injections of calomel, and after the fifth day a notable improvement began. After 4 injections a complete cure had taken place. Upon the same occasion Fournier, Besnier, and Brocq related cases in which the same method of treatment had proved highly successful. The chief objection to it is the pain which follows the injection.

**Biiodosalicylate of Mercury in Syphilis.**—Froloff[3] has employed this salt of mercury in the treatment of 300 cases of syphilis. It contains 20.45% of mercury and 52% of iodin, and occurs as a fine yellow powder, insoluble in water, alcohol, and ether. Mixed with liquid vaselin it does not deposit, but remains suspended. A 10% emulsion in liquid vaselin of biiodosalicylate of soda has also been employed. Injections were made deep into the buttock behind the great trochanter either twice a week or every 4 days. In the beginning of treatment only 6 or 8 divisions of the syringe were injected, but later a whole syringeful was given at each injection. The results were, upon the whole, favorable; the primitive roseola disappeared after 3 or 4 injections; mucous plaques after 4 or 5; primitive scleroses after 7 or 8. Secondary and tertiary symptoms also disappeared under this treatment. Irritation of the gums has been rare and but little marked. The injections have been well borne by weak and exhausted patients. Local irritation after the injections has been quite as frequent as after other mercurials. With this mode of treatment the disease has been rapidly cured.

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[1] Phila. Med. Jour., May 26, 1900.

[2] Gaz. hebdom. de méd. et de chir., Jan. 18, 1900.

[3] Medicinsk Obosr, No. 9, 1899; Jour. de Méd. de Paris, Feb. 18, 1900.

**Syphilis Treated by the Intravenous Injection of Mercury Cyanid.**—Chopping[1] claims the following advantages for this method of treatment: Daily observation of the patient, exactness of dosage, and rapid production of the drug and disappearance of serious lesions. It is especially useful after the failure of other methods. Of 84 cases treated in this manner, but 1 failed to show marked improvement. Under ordinary asepsis the injections are made preferably in the veins of the forearm, a rubber tourniquet being applied to the upper part of the arm. Twenty minims of a 1% solution are employed at each injection, which should be made in the direction of the blood current. The injections are painless unless the vein is missed, and should be made every day unless diarrhea occurs, which is rare.

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[1] Lancet, 1899, p. 432; Am. Jour. Med. Sci., Oct., 1899.

# MATERIA MEDICA, EXPERIMENTAL THERAPEUTICS, AND PHARMACOLOGY.

By REYNOLD W. WILCOX, M.D., LL.D., and A. A. STEVENS, A.M., M.D.,
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**Acoin.**—The hydrochlorate of dipara-anisyl-monophenetyl-guanadin has been introduced under the name of acoin as a new local anesthetic. It appears as a white crystalline powder, soluble in 6.7 parts of cold water. Its solutions remain unchanged for a considerable time when kept in the dark. Its solutions have distinct antiseptic properties, but are more or less irritating. Randolph[1] has used it in affections of the eye in the strength of 1 : 100 and 1 : 300, but found it less satisfactory than either cocain or holocain. He also notes that when the eyes are congested acoin does not give satisfactory anesthesia. Darier[2] has employed acoin to render injections of mercury cyanid painless, but found that while it promptly produced anesthesia, it also excited marked edema of the conjunctiva. Carter[3] has repeated Darier's experiments with satisfactory results, the only drawback being the sense of weight and inconvenience from the swelling, which, however, soon passed away. Gomperz[4] has employed acoin in 2% solution for aural and nasal operations. He states that it irritates more than a 10% solution of cocain, that it is inferior in anesthetic power, and, moreover, that it does not contract the blood-vessels as does cocain.

**Adonidin.**—Stern[5] concludes from a study of this drug that while its physiologic action is similar to digitalis, it may be safely administered in those conditions in which digitalis, if given at all, should be administered with the utmost caution. He asserts that in rapidity and permanency of action it surpasses digitalis, caffein, spartein, strophanthus, and convallaria majalis. As a physiologic diuretic it is decidedly inferior to caffein, strophanthus, spartein, and convallarin. Its greatest diuretic force is exhibited in conditions accompanied by dropsy and low arterial tension. There is no instance on record in which adonidin produced lethal effects in man. The dose of the drug varies according to the object desired : $\frac{1}{32}$ of a grain 2 or 3 times a day may often suffice to overcome asthenic conditions ; $\frac{1}{12}$ of a grain 3 or 4 times a day—the dose most frequently employed by the author—corrected arhythmia, stopped precardial pain, and when used hypodermically often relieved dyspnea ; $\frac{1}{6}$ of a grain 3, 4, or 5 times a day influenced edema and promoted diuresis, especially in cases characterized by low arterial tension. The author reports a number of cases of chronic nephritis in which adonidin proved efficacious as a diuretic and a cardiac tonic.

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[1] Oph. Rec., Aug., 1899.

[2] Clin. Ophthal., No. 12, 1899.

[3] Lancet, Oct. 21, 1899.

[4] Therap. Monatsh., 1900, Heft 1, S. 35.

[5] Centralbl. f. Gynäk., June 2, 1900.

**Airol.**—Fraenkel [1] believes applications of airol (bismuth-oxy-iodo-gallate) are useful in preventing the stitch-abscesses which sometimes follow laparotomy despite all precautionary measures. He believes that airol powder and gauze are to be preferred to airol ointment, as the gauze absorbs the secretions better than the ointment. Döderlein [2] speaks highly of Brun's airol ointment for dressing aseptic wounds. After closing the incision he covers it with a thick and broad layer of the ointment, covers that with gauze strips of adhesive plaster, and does not touch it until the twenty-first day, when he removes gauze, ointment, and sutures. With this method, out of 400 cases, 356, or about 90 %, healed satisfactorily. He recommends the ointment not for any antiseptic properties, but for its hygroscopic, drying, immovable, and hermetically sealing characteristics. Stoeckel [3] also speaks favorably of airol ointment and powder as a dressing for wounds, but prefers sterilized kaolin, as it is nonpoisonous, nonirritating, and odorless, and since it has also great capacity for absorbing moisture.

**Alcohol.**—Favorable reports of the antidotal action of alcohol in carbolic-acid poisoning continue to appear. Rodman [4] reports a case of carbolic-acid poisoning in which about 2 ounces of the pure acid had been swallowed. When first seen, the patient was in a state of profound collapse. Four ounces of alcohol were poured into a stomach-tube passed as far as the pharynx. Two or three minutes later the tube was pushed down to the stomach, and the latter was then washed out with warm water and again with diluted alcohol. Within an hour consciousness had returned and the general condition was much improved. In a few days recovery was complete. In an editorial on "Carbolic Acid and its Antidotes" [5] the writer states that of all andidotes for carbolic acid the best-lauded one for promptness in results is alcohol, which was first suggested by Phelps and Powell. The writer has taken pains to look up a large number of reported cases of carbolic-acid poisoning that have occurred in the past, and finds that where no alcohol was given, in cases that were known or reasonably believed to have retained 60 grains or upward of absolute carbolic acid, the termination in nearly every one was fatal. On the other hand, in all cases where alcohol was given, under the same conditions, the patients survived, although the physicians in attendance had no idea that alcohol possessed any specific antidotal action. [The alcohol treatment of carbolic-acid poisoning is certainly a great advance. Success depends largely upon promptness, and this should be strongly insisted upon.]

**Amyl Valerianate.**—Pouchet [6] recommends, on account of its solvent action on cholesterin, the use of amyl valerianate for gall-stones. According to the author, 4½ gm. of this agent will dissolve 1 gm. of cholesterin. The drug may be given in capsules or in emulsion, in doses of from 3 to 12 grains a day. [The proposition should be clearly stated that, having determined the presence of gall-stones, the remedy is not necessarily surgical. This adds to the list of remedies which are reasonably effective in disintegrating gall-stones.]

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[1] Merck's Arch., May, 1900.

[2] Centralbl. f. Gynäk., July 7, 1900.

[3] Centralbl. f. Gynäk., June 9, 1900.

[4] Merck's Arch., Aug., 1900.

[5] Merck's Arch., Dec., 1899.

[6] Jour. de Praticiens, No. 45, 1899.

**Anticholera Serum.**—Powell[1] states that 5778 out of a population of 12,327 were inoculated with Haffkine's serum and 6549 were not. Among those inoculated there were 27 cases of cholera and 14 deaths; among those not inoculated there were 198 cases of cholera and 124 deaths. The mortality was $7\frac{11}{14}\%$ greater among the noninoculated than among the inoculated. Up to 1895 Haffkine used attenuated virus for his work, beginning with a very weak serum and gradually proceeding to a more and more concentrated one, thus requiring two or more inoculations to produce immunity; but since that date he has inoculated 1123 persons with virulent, recently isolated vibrios. In no case has there been any suppuration or other accident.

**Antiphthisic Serum.**—Holmes[2] writes: "After 16 months of constant study of the effects of antiphthisic serum, T. R., I am impressed with the belief that serum therapy is the coming therapy for tuberculosis." In concluding his paper he makes the following observations: (1) The serum treatment gives the best results in incipient cases or those with small areas of infection; (2) patients with well-established tuberculous lesions require a longer time to bring about a cure than has heretofore been thought necessary; (3) a serious mistake is made in dispensing with the use of the serum too soon in any case that improves under its use; (4) all patients who do well under the serum treatment continue to improve after it has been stopped; (5) the number of bacilli in the sputum when other signs of the disease are diminishing is not a true criterion of the condition of the patient; (6) the treatment can be continued in daily doses for a period of 9 months in individual cases with no bad effects; (7) the cases for this treatment should be carefully selected; (8) climate alone as a therapeutic agent in tuberculosis is not reliable. In a subsequent paper[3] Holmes reports 50 cases treated during 21 months with antiphthisic serum. The results were very encouraging. Ambler[4] also reports results with the serum in incipient phthisis. He believes that it is contraindicated in general miliary tuberculosis, in extensive softening, high pulse, marked emaciation, or when there is a hereditary history. He states that relapses are less frequent than when patients are treated with creasote and allied drugs. He has found, however, that syncopal attacks lasting for 1 or 2 minutes follow the injections in about 5% of the patients. Stubbert,[5] in 1898, reported 36 cases of incipient phthisis treated with serum as an auxiliary to climate. Of the 14 cases apparently cured, he has been able to obtain the following subsequent results: Nine of these patients are still apparently perfectly well; they have no cough, no physical signs, and they are able to attend to their various occupations. He also reports 13 new cases, which have been out of the institution only for a year. These all show persistent improvement. He gives the following summary of all his figures: Of the patients out of the institution for 3 years, 11 % have remained cured; out for 2 years, 14 %; and out for 1 year, 69 %. He believes that antitubercle serum produces some immunity, and that it is a valuable auxiliary to climatic influence. [No remedy nor climate should be exclusively relied upon. When the tuberculous individual is studied with the same care as patients suffering from other infectious diseases, the mortality percentage will rapidly diminish.]

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[1] Jour. Tropical Med., No. 2, 1899.

[2] N. Y. Med. Jour., April 8, 1899.

[3] Jour. Am. Med. Assoc., vol. XXXIII, 1899.

[4] Jour. Am. Med. Assoc., July 8, 1899.

[5] Med. News, Aug. 18, 1900.

**Antipneumococcic Serum.**—McFarland and Lincoln [1] state concerning this treatment that in the few observations that have been made by other writers varying success is reported, but enough cases have been treated to give a very hopeful outlook for the future of serum therapy. The most important observations made are as follows: De Renzi, in 1895 and 1896, studied, comparatively, 13 cases of pneumonia treated without the serum, with 3 (23 %) deaths, and 14 cases treated with the serum, with 2 (14 %) deaths. In the latter series one was *in articulo mortis* when treatment was begun, and the other had renal and arterial disease apart from the pneumonia. Cooke treated 2 patients, Spurnell and Fanoni 1 each, with antipneumococcic serum from different sources with good effect and ultimate recovery. Pane treated 9 cases with an average of 20 cc. of antipneumococcic serum each day. All these recovered except one, in which the treatment was begun late and an insufficient quantity of the serum administered.

Washbourne treated 6 patients, some of them very severe cases, with his serum, and had excellent results, all his cases recovering.

Fanoni [2] writes that the efficiency of Pane's serum in lobar pneumonia has been recognized by impartial observers, such as de Renzi, Maragliano, Cantieri, and Massalongo. He has used this serum with marked success in 18 cases of pneumonia, 4 of which were in children under the age of 3 years. Of these 18 cases only 1 died. The fatal case was a patient in the preagonal stage. In the 4 cases of pneumonia in children there was no doubt as to the efficiency of the serum. All these children recovered after a few days' treatment. Of the last 2 cases, one was 18 months old, and received 4 injections, and the other was 2 years old, and received 3 injections. This serum, when injected early enough, in sufficient quantity (40 cc. of No. 2, daily), and if not deteriorated by age, quickly produces a lowering of the temperature, and an improvement in the subjective comfort of the patient, as well as an amelioration of all the other symptoms. Resolution also tends to take place more rapidly. Canby [3] reports 3 severe cases of pneumonia treated with Pane's serum, in which defervescence occurred in the first two on the second day, and in the third on the fourth day. The dose was 2½ drams, repeated on the following day when necessary. Notwithstanding these favorable reports, many observers, recognizing the natural tendency of the disease toward recovery, are still skeptical as to the therapeutic value of the serum. Thus, Lambert [1] states that the serum is not bactericidal, but bacteriolytic, and can not by itself stop the infective process. The serum he used had been proved in the laboratory to have a definite protective influence, yet in the 12 cases in which he employed it clinically the results were not very striking. It neither produced a crisis nor cut short the pneumonic process in any case. He admits, however, that in certain cases it seems to prevent a general pneumococcus septicemia. Wilson [2] reports 18 cases in which he employed the serum. After the injections the temperature became lower, the pulse slower, the pain less, and the patient felt better, but the duration of the attack did not seem to be lessened or the defervescence hastened. Four of these patients died; while in a series of 20 cases treated without serum, 4 died.

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[1] Jour. Am. Med. Assoc., Dec. 16, 1899.

[2] Pediatrics, May 15, 1900.

[3] Maryland Med. Jour., vol. XLIII, 1900.

**Antipyrin.**—Francini [3] reports a case of menstrual epilepsy in which he had used the three bromids without result. Commencing a few days before the expected period, he administered antipyrin (5 grains) 3 or 4 times daily. For the first time in 5 years this period passed without symptoms, and the treatment continued to prove successful at subsequent periods.

**Antistreptococcus Serum.**—Although many favorable reports of the use of antistreptococcus serum in septic affections continue to appear, it can not be claimed, as in the case of antidiphtheric serum, that its efficacy is absolutely proved. Good results from the use of the serum have been recorded by Arthur,[4] Bruce,[5] Deardorff,[6] Mahon,[7] Galloway,[8] Wood,[9] Bateman,[10] Anderson,[11] Gervis,[12] Hamilton,[13] Maurice,[14] and Rosenthal.[15] Stevens,[16] in discussing the treatment of septic diseases of the puerperium, concludes that statistics do not show that use of the serum has had any very marked influence on the mortality of the disease. There are, however, plenty of cases on record in which good results are claimed to have been obtained by its use. A few facts are quite certain about it: it is useful only in undoubted cases of streptococcal infection; so before using the serum an accurate bacteriologic examination must be made. Also, there appear to be varieties among streptococci, the infections of some of which will be influenced by a peculiar antistreptococcic serum. But all streptococcal infections will not be improved by the serum treatment. The only guide that at present can be given is that, given a case of real streptococcal infection, as shown by an examination of the uterine discharges (without admixture with vaginal), the serum should be tried, and that a large dose should be given at first, to be followed by smaller doses if any benefit results.

[1] Phila. Med. Jour., Mar. 31, 1900.
[2] Phila. Med. Jour., June 9, 1900.
[3] Gaz. degli Osped. e delle Clin., No. 34, 1899.
[4] Brit. Med. Jour., July 8, 1899.
[5] Brit. Med. Jour., July 8, 1899.
[6] N. Y. Med. Jour., No. 1069, 1899.
[7] Brit. Med. Jour., No. 2055, 1900.
[8] Phila. Med. Jour., Aug. 4, 1900.
[9] Lancet, Aug. 11, 1900.
[10] Edinb. Med. Jour., July, 1900.
[11] Brit. Med. Jour., Feb. 17, 1900.
[12] Brit. Med. Jour., May 26, 1900.
[13] Australas. Med. Gaz., Aug. 21, 1899.
[14] Lancet, Aug. 12, 1899.
[15] Phila. Med. Jour., July 22, 1899.
[16] Treatment, Aug., 1900.

The serum can do no harm, and if it does no other good, it acts as an excellent food. Harrison [1] and Anderson [2] speak favorably of the use of antistreptococcus serum in **erysipelas.** Cahall [3] reports a very severe case of **gangrenous stomatitis** in which antistreptococcus serum gave excellent results. Acting on the belief formulated by Hunter, that **pernicious anemia** is due to an infection of the gastric mucosa, Elder [4] and De Witt [5] have used antistreptococcus serum in this disease. Remarkable results are claimed for the remedy. In De Witt's case examination of the blood showed 4000 white, and less than 1,000,000 red corpuscles per cubic millimeter, and 30 % of hemoglobin. Eight injections of 8 cc. each were given at intervals of 2 or 3 days. Three days after the last injection the blood contained 5000 white and 4,960,000 red corpuscles, and 90 % of hemoglobin. In Elder's case the red corpuscles rose in 7 weeks from 797,500 per cubic millimeter to 4,800,000.

**Antitoxin of Diphtheria.**—The reports which have appeared during the past year confirm with remarkable unanimity the value of antitoxin in diphtheria. Siegert [6] quotes statistics of 42,000 cases of diphtheria (not requiring operation) collected from 79 hospitals in Germany, Austria, Hungary, and Switzerland. Of these cases, the average mortality percentage of the 4 years preceding the use of antitoxin (1890–1894) was 41.4, while the average mortality percentage for the 4 years succeeding the use of antitoxin (1894–1898) was 16.5. In the operative cases the average mortality for the preserum period was 60.38 % ; and for the postserum period, 36.32 %. From the two series of comparisons just summarized the author draws the striking conclusion that of 40 ordinary cases of diphtheria which would die under other modes of treatment, 25 are surely saved by antitoxin ; and that of the severest operative cases, the same remedy rescues 24 out of every 60 that would otherwise perish. In view of these facts he argues that it is impossible to hold guiltless the physician who fails to employ this powerful weapon in combating so dangerous a disease. Turner,[7] of the Children's Hospital, Brisbane, Australia, compares the results obtained in 303 cases of diphtheria treated in the preantitoxin period with the results obtained in 317 cases treated in the antitoxin period. The mortality of the former was 42.2 %, while that of the latter was only 12.6 %. Among the laryngeal cases the mortality was reduced by the antitoxin from 59.2 % to 18.6 %. Marsden [8] reports 105 cases of diphtheria treated with antitoxin. In more than half of the cases the antitoxin was injected during the first day, and in the majority of cases during the first 2 days. There was but one death in the series. The membrane disappeared in 83 out of 99 cases within the first 4 days. Laryngeal symptoms were present in 6 cases. Postdiphtheric paralysis was seen in 7 out of 104 cases that recovered. An erythema followed the injection in 20 cases. The author believes that antitoxin may have an irritant effect upon the kidneys, yet this is by no means the rule, and in many cases the action is only temporary. The amount of antitoxin administered varied from 750 to 14,000 units. Wright[1] states that during the last 5 years he has treated with antitoxin 268 cases of diphtheria. Of these 268 persons, 209 recovered and 59 died—a mortality of 22%. That the percentage of recoveries was not larger he attributes to the fact that the larger part of the cases were seen in consultation only when they had been in progress several days and their condition was considered unfavorable. Of those to whom antitoxin was given upon the first day, 12 died and 99 recovered, an approximate mortality of 10%; on the second day, 9 died and 58 recovered—15% mortality; of those after the second day, 37 died and 51 recovered, making a death-rate of these of over 72%, or a mortality of 11.8% of those to whom antitoxin was given within the first 48 hours of disease. Of 121 laryngeal cases, 68 were intubated, 43 of whom recovered. The disease was in the nose in 30 cases; and 17, or over 50%, of these proved fatal. Wright states that he is not influenced in dosage by the age or size of patient, but by the physical condition, extent, and locálization of the membrane, never using less than 1500 units and seldom more than 3000 units. Gagnoni[2] reports 3 cases of grave diphtheria complicated with croup, with symptoms of imminent suffocation, which he treated by intravenous injections of antidiphtheric serum. The results were most satisfactory; the temperature rapidly fell to normal, and the stenotic symptoms disappeared with the expulsion of the false membrane.

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[1] Brit. Med. Jour., July 17, 1900.
[2] Brit. Med. Jour., Feb. 17, 1900.
[3] Phila. Med. Jour., Feb. 17, 1900.
[4] Lancet, No. 1, 1900.
[5] Merck's Arch., Aug., 1900.
[6] Jahrb. f. Kinderh., LII. p. 56:
[7] Brit. Med. Jour., No. 2035, 1899.
[8] Brit. Med. Jour., Sept. 29, 1900.

Other statistical articles bearing on the serum treatment of diphtheria have been written by Billings,[3] Park,[4] Anderson,[5] and Shurly.[6] Zagato[7] confirms the statements previously made by Fedele,[8] Zahorsky,[9] and Fischer[10] of the efficacy of antitoxin when administered by the mouth. It is stated that the serum does not disturb the stomach, but that absorption takes place considerably more slowly (24 to 36 hours) than by the subcutaneous method. The dose recommended by Zagato is 1000 units in milk.

Villy[11] makes a plea for larger doses of antitoxin than are usually employed. He states that in the Asylums Board hospitals the following doses are employed: An ordinary mild case, seen on the first day of the disease, would receive a dose of 2000 units; but when, as sometimes happens, the symptoms have progressed very rapidly, even in a few hours, a larger amount is given. In severe cases it is the rule to give from 8000 to 12,000 units when the patient is first seen, followed by another 2000 to 8000 units every 12 hours for the next 24 or 48 hours, or longer, according to the gravity of the case and the persistence of the exudation. It may be said that antitoxin should be used as early as possible in sufficient dose to produce a distinct reaction in 12 hours. If such reaction is not obtained, a second or third dose must be given, until the effect is apparent. It is best to err by giving more than is necessary rather than by giving too little. There can be no question that a large initial dose will frequently produce surprising effects, even in severe cases which have come under treatment only late in the disease, such cases being but little affected by the usual doses of from 2000 to 4000 units.

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[1] Pediatrics, Mar. 15, 1900.

[2] Ann. de Méd. et Chir. Infant., Aug. 15, 1899.

[3] N. Y. Med. Jour., Feb. 17, 1900.

[4] Med. News, April 14, 1900.

[5] Quart. Med. Jour., vol. VIII, 1900.

[6] Jour. Am. Med. Assoc., May 19, 1900.

[7] Klin.-therap. Woch., No. 12, 1899.

[8] Gaz. degli Osped., Dec. 25, 1898.

[9] Arch. de Méd. des Enfants, Dec , 1898.

[10] Pediatrics, July 15, 1899.

[11] Med. Chron., Jan., 1899.

Musser [1] prefers to give antitoxin in small doses, and, when necessary, at frequent intervals, rather than to follow the tendency of the time to increase the initial amount. The results appear to be as good as any reported, and he has had but one case of erythema and albuminuria and none of the general joint affections. Thirteen cases are reported, all of which terminated in recovery. The author considers that 5 of these were seriously ill and the prognosis doubtful had not the antitoxin been used. In 7 instances the temperature fell to normal, and remained so, in 48 hours. In 5 cases it was normal within 3 days, and in two very severe infections within 5 days. The method is as follows: For children up to 6 or 8 years the initial dose is 500 immunizing units, to be repeated at intervals of 6 hours if the fever does not fall, if the strength of the patient does not improve, or if the local manifestations are spreading. For children over 8 years 1000 immunizing units are given as an initial dose, and this is repeated at intervals of from 8 to 12 hours, as needed. The only disadvantage in this method is the pain caused by the successive injections, but the author has not found that this objection was sufficient to overbalance the discomfort of the urticaria and the general symptoms that appear to be more common after larger dosing.

Ransom [2] has investigated the relation of toxin, alone and together with antitoxin, to diphtheric paralysis. His conclusions are: (1) Paralysis may certainly be expected after intoxication with not less than one-fourth of the minimal fatal dose of toxin; it may occur with doses between one-fourth and one-eighth, but not when the dose is below one-eighth. (2) Antitoxin given 15 to 22 hours after intoxication, with doses not greater than the lethal dose, exercises in large doses a modifying influence on the subsequent paralysis. Small doses of antitoxin have no evident effect in diminishing the paralysis. (3) Transferring these results to practice among human beings, we may expect liberal doses of antitoxin, given early in the illness, to influence favorably the subsequent paralysis, and this beneficial influence is likely to manifest itself, not so much on the local paralysis (soft palate, etc.), as on such symptoms as failure of the heart. Severe cases are, however, likely to be followed by some paralysis in spite of even large doses of antitoxin.

**Antitoxin of Tetanus.**—It is still impossible to form a definite opinion of the value of antitoxin in the treatment of tetanus. During the past year favorable results from its subcutaneous use have been reported by

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[1] Univ. Med. Mag., Mar., 1900.

[2] Jour. of Path. and Bacteriol., VI, No. 4, 1900.

Kraus,[1] Wise,[2] Tiwary,[3] Holsti [4] (2 cases), Blake [5] (4 cases), Roberts,[6] Taylor,[7] Marshall,[8] Klein [9] (2 cases), Yoanna,[10] Rice,[11] James,[12] Adams,[13] Hibbert,[14] Van Natta,[15] Stucky,[16] and Hobson.[17] Cases in which antitoxin was employed subcutaneously without success have been recorded by Berry,[18] Clark,[19] Möller,[20] Werner,[21] Arneill,[22] Wace,[23] Packard,[24] Kraus [25] (3 cases), Clarke,[26] and Blake.[27] Holsti [28] has studied 124 cases of tetanus with reference to the time after the outbreak of the tetanus at which the antitoxin treatment was begun. He finds that in the first 2 days 49 cases were treated and 69.4% died; between the third and the seventh day 49 cases were treated, and 28.8% were fatal. Later than one week, 26 cases were treated and only 7.7% died. This table, however, has little importance, since it merely indicates that severe cases are brought for treatment much earlier than the mild ones. Holstein further notes that the Italians have had remarkably good results with the serum treatment; all of 23 cases reported having recovered, while the mortality with the serum has varied from 41% to 64.7% in Germany, Austria, Switzerland, and France. The author suspects that this may be due to Tizzoni's serum being much more active than those employed in the north of Europe.

Gimlette,[29] Von Leyden,[30] and Johnson [31] each report a case of tetanus successfully treated with antitoxin administered *intracerebrally*. Collier [32] reports 2 cases treated in the same manner; 1 died and 1 recovered. Abbe [33] reports 9 cases treated with serum. Of these, only 2 would be regarded as in any sense of a mild type, 1 commencing on the tenth, and 1 on the nineteenth, day after injury. The other 7 were of a grave type. Of the 7 grave cases, 5 were so severe that they were deemed worthy of being subjected to the test of trephining. Of these 5, 3 recovered and 2 died. The cases which recovered had their onset on the seventh, ninth, and nineteenth days of incubation; the cases which died, on the eighth and the sixteenth day of incubation. There were 5 of the 9 cases in which one could perceive an apparent effect of the serum treatment. The author concludes that antitetanic serum is a valuable adjunct in the treatment of tetanus, and that the cerebral injection is an advance over the subcutaneous method worthy of extended trial and further study. According to Whitmore,[1] the results at Roosevelt Hospital, New York, have not been so successful. He states that of 9 cases treated by trephining and injecting the serum, 7 died. Zupnik [2] says that a series of experimental and clinical researches has led him to conclude, in spite of 2 serious cases in which intracerebral injections were employed with good results, that further tests of the efficiency of this particular method must be secured to prove that the favorable results in the severe cases that recover under its use are to be attributed to it and not to the other means that are employed at the same time. Gibb [3] reports a case in which recovery from tetanus followed the intracerebral injections, but in which death subsequently resulted from cerebral abscess.

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[1] Zeit. f. klin. Med., Bd. XXXVII, Hefte 3 u. 4.

[2] Brit. Med. Jour., June 9, 1900.

[3] Indian Med. Rec., Nov. 15, 1899.

[4] Zeit. f. klin Med., Bd. XXXVII, Hefte 5 u. 6.

[5] Phila. Med. Jour., June 9, 1900.

[6] Brit. Med. Jour., April 28, 1900.

[7] Med. News, July 8, 1899.

[8] Lancet, April 22, 1899.

[9] Deut. med. Woch., Jan. 12, 1899.

[10] Med. Rec., July 29, 1899.

[11] Lancet, Oct. 14, 1899.

[12] Med. Rec., Sept. 9, 1899.

[13] Phila. Med. Jour., Dec. 30, 1899.

[14] New Eng. Med. Monthly, Nov., 1899.

[15] Bull. Gén. de Therap., CXXXIX, 1899.

[16] Merck's Arch., July, 1900.

[17] Merck's Arch., July, 1900.

[18] Lancet, April 29, 1899.

[19] Brit. Med. Jour., Jan. 7, 1899.

[20] Münch. med. Woch., Feb. 28, 1899.

[21] Münch. med. Woch., Feb. 28, 1899.

[22] Med. News, April 22, 1899.

[23] Lancet, May 13, 1899.

[24] Phila. Med. Jour., Nov. 18, 1899.

[25] Zeit. f. klin. Med., Bd. XXXVII, Hefte 3 u. 4.

[26] N. Y. Med. Jour., June 16, 1900.

[27] Phila. Med. Jour., June 9, 1900.

[28] Zeit. f. klin. Med., Bd. XXXVII, Hefte 5 u. 6.

[29] Lancet, July 8, 1899.

[30] Berl. klin. Woch., July 17, 1899.

[31] Ann. of Surg., Mar., 1900.

[32] Lancet, May 13, 1899.

[33] Ann. of Surg., Mar., 1900.

**Antivenomous Serum.**—Semple and Lamb [4] assume from a series of experiments that 225 grains of serum are required to neutralize the fatal dose of cobra venom. When snake-bite is treated, soon after it occurs, with an intravenous injection of antivenomous serum, the authors show by experiment that the result is the same as that obtained when done in glass vessels in the laboratory. Disregarding cases where death occurs very rapidly [probably because the venom is injected directly into a vein], death usually takes place after several hours. During the interval the poison is slowly absorbed. If 225 grains or more of serum be injected before the lethal dose can be absorbed, recovery should take place.

Rennie [5] reports the case of a boy 12 years old who was bitten in the little finger while asleep. When first seen, there were complete paralysis of the left side, marked edema, stupor, labored breathing, frothing at the mouth, and an irregular and intermittent pulse. Twelve cubic centimeters of Calmette's antivenene were injected hypodermically and a stimulating enema was administered. While this was being accomplished, breathing ceased and the heart-beats were only 4 a minute. Artificial respiration was commenced and carried on for 20 minutes, by which time the serum had accomplished its work. The boy steadily improved, and in 5 days was convalescent. The case shows that antivenene may prove successful even in most extreme conditions. Another serious case in which recovery followed the use of antivenomous serum is reported by Beveridge.[6] In 1897 Dyer [7] reported 5 cases of **leprosy** treated with Calmette's antivenomous serum. Of these cases, 2 were apparently cured and 2 were distinctly improved. Woodson,[8] acting on Dyer's advice, employed antivenene in a German woman, aged 36, with well-advanced leprosy. The dose varied from 33 cc. at first to 20 cc. toward the close of treatment. After 2 months' treatment there was remarkable improvement.

**Apomorphin.**—Douglas [9] states that he has obtained excellent results from the use of apomorphin as a hypnotic in various forms of **insomnia.** In 300 patients there were but 2 or 3 on whom it had but slight effect. One-thirtieth of a grain is said to be the average dose. The dose, however, should be so adjusted as to be large enough to produce sleep and small enough to avoid nausea. The drug is administered subcutaneously. [Different specimens of the drug give different results, thus suggesting the purity of the remedy. Sleep is by no means so constant a sequel as might be anticipated from a reading of the literature.]

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[1] Phila. Med. Jour., June 9, 1900.

[2] Prag. med. Woch., No. 2599, 1899.

[3] Brit. Med. Jour., July 1, 1899.

[4] Brit. Med. Jour., No. 1996, 1899.

[5] Lancet, Nov. 25, 1899.

[6] Brit. Med. Jour., Dec. 23, 1899.

[7] New Orl. M. and S. Jour., Oct., 1897.

[8] Phila. Med. Jour., Dec. 23, 1899.

[9] Merck's Arch., June, 1900.

**Arecolin.**—Clemensha[1] states that the hydrobromate of arecolin, an alkaloid found in the areca nut, is a white, crystalline, soluble salt, which when applied to the eye in the form of a ½% or 1% aqueous solution causes contraction of the pupil. A ½% solution dropped into the conjunctival sac causes burning and slight congestion. In from 3 to 5 minutes the pupil begins to contract, and reaches its maximum in from 10 to 15 minutes, accompanied by spasms of the ciliary muscle. The maximum effect remains for 15 minutes or so, after which the pupil gradually returns to its normal condition, usually in the course of an hour or two. In the normal eye tension is apparently unaffected, but in glaucoma clinical results show this drug to be the equal of eserin. The drug keeps well in solution. According to Frohner, arecolin, when injected hypodermically, is a more powerful sialogog than pilocarpin.

**Argentamin.**—Argentamin, or ethylene-diamin-silver phosphate, is a solution of 10 parts of silver phosphate in a 10% aqueous solution of ethylene-diamin. It is a fluid with an alkaline reaction, which gives no precipitate with chlorids or albumins, but requires to be kept in the dark. It has been claimed that argentamin is superior to silver nitrate in being more antiseptic and in having a greater penetrative power. Lockwood[2] claims for it another superiority over other silver salts in the astringency which it possesses, and which is absent in protargol and largin. Williams[3] states that he has never met any remedy recommended for **gonorrhea** which has given such entire satisfaction as injections of argentamin preceded by injections of hydrogen dioxid. He recommends that only fresh solutions be used, and of a strength not less than 1 : 3000. Imre[4] speaks highly of 5% solutions of argentamin in various inflammatory affections of the conjunctiva. On the other hand, Kopp[5] has found it very irritating and in no way superior to silver nitrate.

**Argentol.**—Argentol, according to Cipriani,[6] is silver oxychinolin sulphate. It is an almost insoluble powder that easily decomposes, when brought in contact with septic substances, into oxychinolin and metallic silver, both of which in the nascent state are powerful antiseptics. Owing to this property, argentol has been recommended as a dusting-powder or an ointment for wounds, syphilitic and simple ulcers, skin diseases, etc., or as an emulsion (1 : 1000 or 1 : 3000) to inject in gonorrhea. Internally Cipriani found it useful as an intestinal antiseptic in conditions due to autointoxication. As it is very slightly toxic, the daily dose may be raised to 15 grains.

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[1] Buffalo Med. Jour., Sept., 1899.

[2] Chicago Med. Rec., April, 1900.

[3] Memphis Med. Monthly, July, 1899.

[4] Therapist, July 6, 1900.

[5] Münch. med. Woch., Aug. 1, 1899.

[6] Allg. med. Central. Zeit., Aug. 26, 1899.

**Argonin L.**—This is a combination of silver (10%) with a sodium compound of casein. It is a fine, light yellow powder, readily soluble in cold water, and the solution keeps for months. Injections of a 1% solution, according to Jellinek,[1] have proved very useful in **gonorrhea.**

**Arsenic.**—Ewald [2] confirms Mabille's observation that arsenic obviates the unpleasant symptoms excited by thyroid preparations. In 5 cases of idiopathic goiter, in a case of obesity, and in 1 of infantile myxedema, iodothyrin was given in progressive doses of from $3\frac{4}{5}$ to 30 or $38\frac{1}{2}$ grains daily. At the same time arsenic was given, either in pills or as Fowler's solution, in doses, increasing proportionately to the iodothyrin, of $\frac{1}{64}$ to $\frac{1}{10}$ or even $\frac{1}{8}$ grain daily. The results fully confirmed Mabille's experience, for though the 7 cases took respectively 231, 111, 86, 320, 108, 296, and 125 iodothyrin tabloids, containing nearly 4 grains each, beyond occasional increased frequency of the pulse no symptoms of thyroidism appeared, so that the course could be continued uninterruptedly. Arsenic, therefore, appears to suppress thyroidism with greater certainty than atropin does iodism, and it is now possible to give iodothyrin safely in doses and for a period capable of producing definite therapeutic effects. Railton [3] reports 4 cases of neuritis following the use of arsenic. The first case was a girl, aged 12 years; 10 drops of liquor arsenicalis were given 3 times a day for 3 weeks, after which time recovery was complete. In all, $6\frac{3}{10}$ grains of arsenous acid were taken. Two weeks later the legs became weak, and soon all the symptoms of peripheral neuritis developed. The second case, a girl, aged 11 years, took in 4 weeks $8\frac{2}{5}$ grains of arsenous acid. The third case took $8\frac{1}{10}$ grains in 3 weeks, and the fourth $9\frac{1}{10}$ grains in 24 days. In all, the symptoms of paralysis appeared in from 1 to 3 weeks after the drug had been discontinued. They all made good recoveries, but convalescence was prolonged. It is impossible to tell in any given case whether the treatment is going to be followed by toxic neuritis or not. The author would strongly urge that no aggregate dose amounting to more than 4 grains of arsenous acid be administered to a child during an attack of chorea.

**Asparagus.**—Hare [4] reports 3 cases in which he used the fluid extract of asparagus as a diuretic. The first case was a patient with cirrhosis of the liver, who passed during a period of 15 days, on an average, between 35 and 48 ounces of urine a day. After the administration of the fluid extract of asparagus in doses of a dram, thrice daily, the quantity of urine for 12 days was maintained at an average of between 55 and 65 ounces. On withdrawal of the drug the quantity of urine gradually fell to between 35 and 45 ounces. Under the treatment the dropsy disappeared. In a case of double mitral disease with edema, in which only 20 to 25 ounces of urine were passed in 24 hours, and in which digitalis, potassium bitartrate, and infusion of juniper berries gave no increase, the asparagus caused an increase in the quantity of urine from 27 to 40 ounces. In a case of advanced atheroma, with aortitis, and probably fatty heart, no marked effect was produced by the asparagus.

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[1] Wien. med. Woch., p. 209, 1899.

[2] Die Therap. d. Gegenw., Sept., 1899.

[3] Med. Chron., Feb., 1900.

[4] Therap. Gaz., Sept. 15, 1899.

**Aspirin.**—This compound is produced by the action of acetic anhydrid on salicylic acid. It appears as small, colorless, tasteless, crystalline needles, which are almost insoluble in cold water. The advantage claimed for aspirin over other salicylic compounds is its freedom from irritating properties, which is due to the fact that it is not decomposed in acid gastric juice, but in the alkaline juices in the intestine. It may be given in the form of powders, in doses of 15 grains 3 or 4 times a day. Among those who have praised its virtues in rheumatism and kindred affections may be mentioned Witthauer,[1] Rénon,[2] Liesau,[3] Zimmerman,[4] Dengel,[5] Floeckinger,[6] Friedeberg,[7] and Ketly.[8]

**Belladonna.**—Hodghead[9] reports 30 cases of bronchopneumonia in children with only 2 deaths. These good results he attributes to the use of belladonna, administered in doses of 2 drops, every hour or two until the desired effect is obtained. The author concludes that calomel should be used in small doses until a cathartic effect is secured; that belladonna in small doses is mildly narcotic, and brings about a condition more comfortable to the child; that it is a heart tonic; that it is a respiratory stimulant; and that it produces dilation of the superficial capillaries and relieves the congested lungs. The most important influence, however, is that it diminishes the secretion in the bronchi and the pulmonary tissues. The drug is not so effective in the beginning of the disease as it is later, when bronchial secretion is abundant. Coutts[10] also calls attention to the value of large doses of belladonna in the bronchopneumonia of childhood.

**Bromoform.**—Favorable reports of the use of bromoform in whooping-cough have become so numerous that the drug must be assigned an important place in therapy of this disease. Cases of poisoning which appear now and then, however, indicate that considerable caution must be exercised in its administration. The trouble has not been so much with the drug itself as with the dose. In the YEAR-BOOK for 1900 we drew attention to the fact that as bromoform is heavier than either water or mucilage, its suspension in the latter can only be temporary, and failure to shake the bottle before each administration may lead to serious consequences. Cases of poisoning have been reported by Stone,[11] Stokes[12] (2 cases), and Darling.[13] All of these recovered under stimulating treatment. Darling's case was a girl of 6 years, who took 1½ drams because she liked the taste of it. The symp-

[1] Die Heilkunde, No. 7, 1899.

[2] La Semaine méd., June 27, 1900.

[3] Deut. med. Woch., May 24, 1900.

[4] Berl. klin. Woch., July 2, 1900.

[5] Berl. klin. Woch., July 2, 1900.

[6] Med. News, Nov. 18, 1899.

[7] Centralbl. f. innere Med., April 14, 1900.

[8] Die Heilkunde, Oct., 1899.

[9] Pediatrics, Sept. 1, 1899.

[10] Brit. Med. Jour., No. 1987, 1899.

[11] Boston M. and S. Jour., Feb. 16, 1899.

[12] Brit. Med. Jour., May 26, 1900.

[13] Brit. Med. Jour., June 2, 1900.

toms were unconsciousness; slow, shallow respirations; an extremely feeble, rapid pulse; contracted pupils; and cyanosis. Lavage with sodium bicarbonate solution, then Condy's fluid, followed by strong coffee and ammonia both by tube and *per rectum*, were the efficient means of treatment. In Stone's case the patient had been pronounced dead of "black diphtheria" by a physician who had been called in previously. Somerville,[1] having found the usual methods of prescribing bromoform unsatisfactory, proposes the use of glycerin as a solvent. A most satisfactory formula, which has stood the test of time, is said to be the following: Bromoform, 2.0 cc.; 90% alcohol, 7.5 cc.; glycerin sufficient to make 30.0 cc. This solution has the advantage of mixing well with water, and many other drugs can be added without throwing the bromoform out of solution. Flavoring agents may also be added.

**Bromopin.**—This is a combination of bromin (10%) with oil of sesame. Certain observers, including Schulze,[2] Zimmerman,[3] and Wulff,[4] have recommended it in epilepsy and other nervous affections, claiming for it freedom from the disagreeable effects which often attend the exhibition of the alkaline bromids.

**Calcium Eosolate.**—This a derivative of creasote, of which it is said to contain about 25%. It appears as a grayish powder, of a slightly acid taste, and soluble in about 10 parts of cold water. According to Stern,[5] it has proved valuable in **diabetes insipidus, diabetes mellitus,** and **phthisis.** The dose is from 4 to 10 grains, 3 or 4 times a day. Five cases of diabetes mellitus under treatment with calcium eosolate and a milk diet ceased excreting dextrose, gained in weight, and improved otherwise.

**Camphor.**—Alexander[6] strongly recommends the subcutaneous use of camphor ($\frac{1}{6}$ to $\frac{1}{3}$ of a grain) in the form of camphorated oil in the treatment of **pulmonary tuberculosis.** On the other hand, Criegern[7] finds that it has no advantages over the other balsamic preparations, and that it is distinctly contraindicated in nephritis and when there is tendency to hemorrhage.

**Cantharides.**—Henderson[8] reports a case of **hematuria** of $2\frac{1}{2}$ years' duration which was promptly cured by the daily administration of 5-minim doses of tincture of cantharides. Other hemostatics had been employed without avail. The author attributes the good results to the tonic effect of the drug on the kidney.

**Carbolic Acid.**—Baccelli[9] states that he has treated 40 cases of tetanus with subcutaneous injections of carbolic acid, and of these, only one died. The details to be observed are: Subcutaneous administration in dose of from $\frac{1}{3}$ to $\frac{2}{3}$ of a grain every 2 or 3 hours until a daily amount of from 6 to 12 grains is reached. Nietert and Amyx[10] report 4 cases of tetanus treated by Baccelli's method. Of these cases, 3 died. The first received during 84 hours 21½ grains of carbolic acid; the second, 11¾ grains in 30¼ hours; the third, 8 grains in 16 hours; and the fourth (recovery) received 93 injections of a 10% solution, and a total of 267 grains in 8 days. The last patient received 99 grains of carbolic acid in the first 24 hours, without untoward effects. Hanson,[1] Bussi,[2] Belfield,[3] Woods,[4] and Pinna[5] also report single instances of cured traumatic tetanus treated by this method. The last-named observer in commenting upon his case cites statistics compiled by Ascoli which are as follows: Baccelli's method, 1 death and 32 recoveries; Tizzoni's serum, 11 deaths and 36 recoveries; Behring's serum, 13 deaths and 20 recoveries.

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[1] Squibb's Ephemeris, 1900.

[2] Inaug. Dissert., Göttingen, 1899.

[3] Neurol. Centralbl., No. 11, 1899.

[4] Aerzt. Monat., No. 11, 1899.

[5] Merck's Arch., Mar., 1900.

[6] Münch. med. Woch., XLVII, No. 9.

[7] Berl. klin. Woch., Oct. 23, 1899.

[8] Indian Med. Gaz., vol. XXXIV, No. 118, 1899.

[9] Riforma Med., vol. III, p. 315, 1899.

[10] St. Louis Med. Rev., Dec. 30, 1899.

Bell[6] reports a case of **bubonic plague** in a man, aged 24, that recovered under the use of carbolic acid (12 grains in solution every 3 hours). He had all the characteristic symptoms, his temperature running to 106.4° F. A similar case is reported in "Merck's Archives" for January, 1900.

Amadori[7] reports a case of **malignant edema** in which injections of carbolic acid were used successfully. Seven injections of ½ of a grain each were made into the swelling, and on the next day 6 more injections of ⅓ of a grain each were given. Recovery was complete within 5 days.

Mackenzie[8] calls attention again to the fact, established by the researches of Scheurlen and Bechmen, that the germicidal action of carbolic acid is increased by the addition of sodium chlorid. These observers found that a 1% solution of carbolic acid in water failed to destroy Staphylococcus pyogenes aureus in 5 minutes, but a solution of the same strength, with 24% of common salt, destroyed the same organisms in 1 minute. They also found that 1% of carbolic acid with 12% and with 20% of salt destroyed anthrax spores in 3 days at latest, while this solution without the addition of salt had hardly any effect upon the spores. The germicidal effect of cresols was also enhanced by the addition of salt.

Harrington[9] calls attention to the fact, of which many physicians are ignorant, that dilute solutions of carbolic acid applied to the extremities for a number of hours may produce gangrene and total destruction of the part. Eighteen cases of gangrene from this cause have come under the author's personal observation, and he has been able to collect 114 additional cases from literature. In most of the cases the solutions did not exceed in strength 5% of carbolic acid. It is evident that carbolic acid solution in any strength applied as a moist dressing is dangerous, and ought never to be used. The fact that it is often used without bad results renders it the more dangerous.

**Carbon Dioxid Gas.**—Norton[10] reports a series of 150 cases of **whooping-cough** treated by the rectal administration of carbon dioxid gas. Of the total number, 143 were decidedly benefited. The vomiting speedily ceased, and the paroxysms became less frequent and severe. The duration of the disease was not influenced. In another series of 20 cases in which the gas used was not nascent, but had been stored in tanks, the result was absolutely negative.

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[1] Cleveland Med. Gaz., Oct., 1899.

[2] Gaz. degli Osped. e delle Clin., Sept. 24, 1899.

[3] Chicago Med. Recorder, April, 1900.

[4] N. Y. Med. Jour., Sept. 9, 1899.

[5] Il Policlinico, No. 17, 1899.

[6] Lancet, July 7, 1900.

[7] Gaz. degli Osped. e delle Clin., No. 67, 1899.

[8] Public Health, May, 1900.

[9] Am. Jour. Med. Sci., July, 1900.

[10] Arch. of Ped., April, 1900.

**Chaulmoogra Oil.**—Savill [1] reports a case of leprosy of 4 years' duration which was much benefited by chaulmoogra oil. The remedy was given in doses of 12 drops daily, with persistent increase, so that by the end of a year the daily amount had become 400 minims. No untoward symptoms resulted; on the contrary, under the influence of the treatment the anesthesia, swelling, and erythema had entirely disappeared.

**Chinocol.**—Kossman and Zander [2] from a series of experiments conclude that a 20% solution of chinocol is the best disinfectant for common use in obstetric practice. Its superior efficiency in destroying germs and its freedom from irritating properties make it a satisfactory disinfectant for the hands.

**Chloreton.**—The therapeutic properties of chloreton, which is trichlor-tertiary-butyl-alcohol, were brought to the attention of the profession last year by Houghton and Aldrich.[3] The authors recommended it especially as a local anesthetic and as a hypnotic. Dewar [4] reports 5 cases in which he found chloreton very efficient as a local anesthetic and antiseptic. The case was that of a man the skin of whose arm had been drawn between a pair of cog-wheels, causing a serious laceration, which extended from the wrist to the shoulder. The wound was bathed with a saturated aqueous solution of chloreton, 30 stitches were introduced without pain, and the parts were dusted with chloreton crystals and subsequently covered with a dressing of chloreton gauze. The wounds healed promptly without sloughing or discharge. Rudolph,[5] on the other hand, has formed an unfavorable opinion of chloreton. He believes that it is inferior to cocain as a local anesthetic. When given internally to animals, he finds that it has a profoundly depressing effect on the temperature, and in consequence he urges great caution in using it in medical practice. To prove that chloreton is a safe hypnotic, Donald [6] cites the case of a morphin habitué who took in one day 120 grains of the drug, and in consequence slept almost continuously for 6 days, when he awoke without bad effects. Wade [7] is enthusiastic over the value of chloreton in the treatment of the insane. He reports 18 cases of various conditions in which it was used as a hypnotic with advantage. The average dose was 35 grains. He believes that the drug exerts some curative influence in cases of mania and agitated melancholia. Hill [8] believes that chloreton is an ideal hypnotic. It does not depress the circulation nor disturb the stomach. He found it particularly useful in delirium tremens and in cases in which morphin had lost its effect. He recommends doses of from 15 to 20 grains. Wilcox [9] believes that chloreton is our closest approximation to that theoretic hypnotic of which we have been in search, and recommends it for its safety, reliability, and ease of taking. Morton [1] comments favorably on the use of chloreton in ophthalmic practice. In inflammatory conditions of the eye he prefers it to boric acid as being more antiseptic and more sedative. Its combination with cocain markedly prolongs the anesthetic effect of the latter.

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[1] Lancet, vol. I, 1900.

[2] Centralbl. f. Gynäk., June 2, 1900.

[3] Jour. Am. Med. Assoc., Sept. 23, 1899.

[4] Therap. Gaz., Feb., 1900.

[5] Canad. Pract. and Rev., June, 1900.

[6] Therap. Gaz., Jan. 15, 1900.

[7] Jour. of Ment. and Nerv. Dis., Aug., 1900.

[8] N. Y. Med. Jour., Aug. 18, 1900.

[9] Med. News, April 14, 1900.

**Chloroform.**—Bose [2] states that he has obtained excellent results in trigeminal neuralgia and severe headache from the introduction just inside of the external auditory meatus of pledgets of cotton-wool well soaked in chloroform. The pledgets are covered with dry cotton-wool before being inserted. Within half a minute a burning sensation is excited, and as this increases the headache disappears. Whyte [3] reports 6 cases of **tapeworm** treated with chloroform; of these, only one was completely successful; one other was doubtful, as the patient could not be traced. The dose varied from ½ to 1 dram. A dose of 1 dram produced rapidly in most cases a comfortable sleep of about an hour's duration. Leichtenstern [4] gives his experience with chloroform as an anthelmintic. The parasite was Tænia saginata. Out of 13 cases only one proved successful; the others were almost all treated subsequently with success by the administration of fluid extract of male-fern in dose of 2 to 2½ drams. The chloroform was given to the extent of 1 dram, usually in 2 or 3 portions at intervals of 1 or 2 hours. In most of the cases there was no disagreeable effect, apart from occasional headache, giddiness, or sickness, such as might be expected with any anthelmintic. In others a very deep sleep occurred, lasting in one case for 24 hours; in 2 cases such severe collapse occurred that there was considerable anxiety for the patient's life. The conclusion is that chloroform is of no use in the treatment of Tænia saginata. As for Tænia solium, while he will not deny that it may be as efficacious as some allege, he thinks that less dangerous remedies, such as kousso, should be employed.

**Cimicifuga.**—Mendel and Robin [5] draw attention to the use of cimicifuga in tinnitus aurium. They claim that after using the fluid extract (10 to 60 drops; the average dose being 30 drops a day) for 2 or 3 days the subjective sounds often completely disappear.

**Cinnamic Acid.**—Hessen [6] reports 47 cases of phthisis in which excellent results are claimed to have been secured by Landerer's method of using cinnamic acid subcutaneously. Fraenkel [7] concludes from a study of the entire literature of the subject and from a series of experiments conducted by himself that Landerer's treatment with cinnamic acid gives no better results than can be obtained by other means. The drug appears to be useless as an inhalation, the results of its administration by the stomach are very doubtful, intravenous injection is excessively painful, and subcutaneous injection equally so, unless cocain be used. The results of 12 cases were that 3 died, 1 became worse, 3 were unchanged, and 5 were improved; of the latter, only 1 to such a degree that any influence on the part of the medicine could be suspected. In a group of 329 cases collected from the literature the proportion of improvements was 65%.

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[1] Merck's Arch., July, 1900.

[2] Indian Med. Rec., Jan. 3, 1900.

[3] Scottish M. and S. Jour., Nov., 1899.

[4] Die Therap. d. Gegenw., Sept., 1899.

[5] Therap. Gaz., Feb., 1900.

[6] Therapist, Mar. 15, 1900.

[7] Deut. Arch. f. klin. Med., Feb. 6, 1900.

**Citric Acid.**—Somers[1] reports a number of cases of atrophic rhinitis in which applications of citric acid proved useful in relieving the ozena. In all the cases no other remedies were applied, except that the parts were cleansed with an alkaline solution, this being followed by hydrogen peroxid, and again with the former solution, and all crusts removed. This was repeated every day, and the patient was instructed to do the same 3 times daily, and to insufflate into the nostrils the following powder: Citric acid, 25 parts; sugar of milk, 75 parts. In from 2 days to 3 weeks the odor in all cases practically disappeared. The author draws the following conclusions: The drug is of great value in preventing the fetid odor of atrophic rhinitis. The successful action depends upon its direct application to the diseased tissues; for this reason the removal of all foreign material is absolutely necessary. After its use the ozena usually remains absent from 1 to 2 days, and in exceptional cases longer, depending upon the extent and severity of the morbid process. It exercises no direct action upon the morbid tissue in the direction of restoration to its normal functions. Unless used at more or less regular intervals its action is but transient, and the ozena speedily becomes prominent again. To a moderate extent it inhibits scab formation. Hamm[2] also speaks highly of citric acid as a deodorizer in atrophic rhinitis. Moncorvo[3] recommends applications of a 10% solution of citric acid to the pharynx and larynx in whooping-cough. These applications are made every 2 hours by means of a fine brush with a curved handle. He claims that under this treatment the attacks rapidly diminish in frequency and intensity, and that a cure is effected in from 15 to 20 days.

**Cocain.**—Since Bier[4] first announced the results of his experiments undertaken to determine the practical utility of rendering large areas of the body anesthetic by injecting cocain into the subdural space of the spinal cord, numerous papers have appeared commenting favorably on this method of inducing anesthesia. Tuffier[5] states that he has operated 63 times with anesthesia induced by this method; the cases including operations on the legs, perineum, rectum, abdomen, and internal and external genito-urinary organs of both men and women. In each case the anesthesia was absolute, and every case made a rapid recovery without complications. A freshly prepared 2% solution of cocain is employed, and sterilized just before it is used. The skin is prepared as for any operation; the needle, the syringe, and the operator's hand being sterilized, the needle is then introduced into the space between the fourth and fifth lumbar vertebræ. The amount of cocain injected should not be more than 0.015 milligram. Anesthesia is absolute in from 4 to 10 minutes, and its duration is from 1 to 1½ hours. In no case has an accident attended the administration of the anesthetic. Occasionally some headache, nausea, and vomiting follow, but they are of short duration. The pupils are usually dilated, there is some benumbing of the limbs, and the pulse is more rapid than is normal, but it generally subsides within 24 hours. In 15 cases an elevation of temperature was observed, and was undoubtedly due to the direct action of the drug on the heat center. [Later observations show that this method is uncertain in producing anesthesia, and accidents are not rare.] Kohn[1] reports a case showing a marked idiosyncrasy toward cocain. A strong man, aged 46, was seized with severe epistaxis, which recurred in spite of treatment. Two applications of a 10% solution of cocain were made by means of a swab of wool to the bleeding spot in the floor of the left nostril. The result was an outbreak of epileptiform convulsions which lasted for nearly 10 minutes.

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[1] Therap. Gaz., Mar. 15, 1900.
[2] Therapist, June 15, 1899.
[3] Jour. de Praticiens, July 8, 1899.
[4] Deut. Zeit. f. Chir., April, 1899.
[5] La Semaine méd., May 16, 1900.

**Codein.**—Lochboeler[2] draws the following conclusions from a study of this drug: Codein is a most valuable drug as a sedative in the various forms of cough. It is an excellent anodyne in the pains of pelvic and abdominal origin where morphin is contraindicated and where the intestinal functions should not be deranged. It is the best remedy in the medicinal treatment of diabetes mellitus. It is best administered in the form of sulphate for internal use, and as a phosphate hypodermically. Poisonous doses produce great restlessness, intense irritation of the entire body, spasms of the muscular system, and generally dilation of the pupils. No fatal case of codein-poisoning is recorded.

**Cod-liver Oil.**—Maigne[3] believes that the only kind of cod-liver oil that should be used is the pure oil, made from absolutely fresh livers that have been carefully washed and preserved in air-tight vessels, or after they have been exposed to a temperature of not over 157° F. Thus prepared, the oil does not leave a disagreeable after-taste that is capable of producing eructations.

**Creasote Carbonate.**—Cornet[4] refers to the advantage which this preparation has over pure creasote when administered in phthisis. He finds that it does not irritate the stomach, but that it often increases the appetite and bodily strength, lessens expectoration, and causes a general improvement in the patient's condition. Creasotal is to be administered to patients 3 times daily in milk, claret, beef-tea, or capsules a quarter of an hour after eating. The beginning dose is 5 drops, to be increased by 2 to 5 drops until about 30 drops are taken at a dose; and the administration is to be continued for from 6 to 8 weeks. Then the remedy should be discontinued for about a week, after which its administration may be begun again. The very large doses of from 2 to 3 teaspoonfuls which Chaumier has recommended frequently render the drug distasteful to the patient and cause digestive disturbance. Pollak[5] also reports favorably upon the use of creasote carbonate in phthisis. Corgier[1] calls attention to the value of creasote carbonate in infections of the respiratory tract other than phthisis. He speaks very favorably of its action in bronchitic croupous pneumonia and bronchopneumonia. [The reports are so harmonious as to the carbonate that it seems quite remarkable that creasote with all its disadvantages was used so long.]

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[1] Med. Rec., Mar. 24, 1900.

[2] Jour. Am. Med. Assoc., Dec. 2, 1899.

[3] Gaz. hebdom. de méd. et de chir., July 22, 1900.

[4] Nothnagel's Spec. Path. u. Ther., vol. XIV, part 3.

[5] Wien. klin. Woch., No. 3, 1900.

**Cresols.**—Seybold[2] concludes from a study of the disinfectant action of the various cresols (metacresol, orthocresol, paracresol, tricresol, and phenol) that metacresol should be recommended in practice because it is more disinfectant than phenol, is not so toxic, and 2% solutions are clear, odorless, and do not injure the hands or instruments.

**Digitalis.**—Arnold and Wood[3] draw the following conclusions from a comparative study of digitalis and its derivatives: Digitalin and digitoxin each represent the full circulatory powers of digitalis. Digitalis, digitalin, and digitoxin stimulate the cardio-inhibitory mechanism both centrally and peripherally. In larger doses they paralyze the intrinsic cardio-inhibitory apparatus. They all cause a rise of blood pressure by stimulating the heart and constricting the vessels. Very large doses paralyze the heart muscle of the mammal, the organ stopping in diastole. Digitalin of Merck is a stable compound, 1 gram of it being equivalent to about 70 cc. of tincture of digitalis. Digitoxin is not to be recommended for human medication on account of its irritant action, which makes it liable to upset the stomach when given by the mouth, or to cause abscesses when given hypodermically, and on account of its insolubility, which renders it slowly absorbed and irregularly eliminated, having a marked tendency to cumulative action. Zeltner[4] concludes from a comparative study of Merck's digitoxin and digitalis leaves that digitoxin in promptness, energy, and duration of action is equal and sometimes superior to the mother plant. The dose employed was $\frac{1}{250}$ of a grain 3 times a day. Potain,[5] in dealing with the selection of suitable cases for digitalis, states that when inequality, irregularity, and insufficiency of the pulsations are not present, or when there is no dropsy of the cellular tissues and serous cavities, contraindications for digitalis exist. The author does not look upon a permanently infrequent pulse as a contraindication. He even considers that many indications of infrequent pulse are caused by the pulsation failing to reach the wrist, and that digitalis may therefore do good. A strong contraindication to digitalis is the presence of myocardial lesions. In these cases, not only is the drug ineffectual, but it may be dangerous. Thus myocarditis, senile cachexia, fatty degeneration, etc., call for the very greatest care in the use of this drug. Potain looks upon aortic incompetence as, generally speaking, a contraindication, unless there is, as in some few cases, undue acceleration of the beats. Dyspepsia very often causes digitalis to disagree, for under these circumstances patients bear it badly. Similarly a cachectic condition is a contraindication. As to the bad results of digitalis, the writer states freely that they are not properly recognized. Some nocturnal delirium is one of the first to appear, and one of the most frequently overlooked. Pallor, coldness of the extremities, trembling, and contraction of the pupils are important indications to suspend the drug. Some patients die suddenly of syncope; others gradually. Death from digitalis is most frequently met in Bright's disease, in arthritic and anemic subjects, and in persons with aortic incompetence or delirium tremens. Occasionally there are curious idiosyncrasies to be noted, such as melancholia and night-terrors. As an unusual result, may be mentioned pulmonary apoplexy, especially when tricuspid incompetence is present. Huchard [1] states that digitalis is especially indicated in simple dilation. It is not contraindicated except in advanced stages of myocardial degeneration. When the myocardium is seriously affected, caffein with sodium benzoate should be given hypodermically. England [2] suggests that the apparent cumulative action of digitalis may be due to the slowness of absorption and of elimination of digitoxin. Bartholow [3] urges more care in the selection of cases for the administration of digitalis. He holds that the view that digitalis is a "heart tonic" suitable for all conditions of weak heart is fallacious. The special conditions justifying its use are mitral lesions, low vascular tension, the absence of obstructive disease of the aortic orifice, general dropsy, and such a state of tolerance of the digestive apparatus that appropriate food can be taken and assimilated. Porter [4] concludes from a study of digitalis that the chemical composition of the drug is complex, some of its active principles antagonizing; that the various preparations of the drug differ widely in their composition and action; that the so-called cumulative action is the result of its contracting the arterioles and so shutting off nutrition; that it is both a useful and a dangerous remedy, but its range of usefulness is very limited; that it is valuable only in lesions of the mitral valve, and then only for a short time, and should be discontinued as soon as these have been overcome; that it is useful as a diuretic only when the arterial tension is low and the kidney is engorged with blood; and that digitalis decreases the excretory action of the normal kidney and impairs its nutritive activity. Bosse [5] speaks favorably of the action of digitalis dialysate. The beginning dose was 20 drops 3 times a day. It is believed to be much more active than other preparations because it is made from the freshly gathered plant. The most noticeable thing was the increase in the amount of urine. In several cases this went rapidly from less than 200 cc. a day to 4000 cc., and in one case to 6000 a day. Lack of cumulative action is attributed to the fact that the dialysate is absorbed rapidly. In only one case was nausea observed. Cutler [6] calls attention to the great advantage of England's fat-free tincture of digitalis, the object being to eliminate the irritating oils. Loomis [7] has treated 10 cases of acute alcoholic delirium with large doses of tincture of digitalis. A half ounce of this was given every 4 hours for 3 doses. There was no marked effect upon the pulse, the chief action apparently being narcotic. The author concludes that the indiscriminate use of these large doses of digitalis is dangerous. They should be restricted to robust patients without complications. [The digitalis question is but slowly approaching solution; we welcome these contributions in the hope that the findings of the laboratory and of the clinic may some time be harmonious.]

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[1] Inaug. Thesis, Montpellier, 1899.

[2] Riforma Med., No. 59, 1899.

[3] Am. Jour. Med. Sci., Aug., 1900.

[4] Münch. med. Woch., June 26, 1900.

[5] Jour. de Méd., April 10, 1900.

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[1] Méd. moderne, Feb. 17, 1900.

[2] Am. Jour. Pharm., Aug., 1899.

[3] Phila. Med. Jour., Nov. 11, 1899.

[4] Phila. Med. Jour., May 12, 1900.

[5] Centralbl. f. innere Med., July 8, 1899.

[6] Boston M. and S. Jour., Sept. 20, 1900.

[7] Med. News, Aug. 18, 1900.

**Dionin.**—Numerous papers have appeared during the past year indicating that we have in dionin (ethyl-morphin hydrochlorate) a valuable substitute for morphin for use as an anodyne, sedative, and hypnotic. The great advantage of dionin over other preparations allied to morphin is its free solubility in water (14 : 100). According to Winternitz,[1] dionin, like codein, has no influence on the respiratory center and does not affect the digestive tract. Higier,[2] Janisch,[3] Kobert,[4] and Smithwick[5] speak enthusiastically of dionin as a sedative in phthisis and other chronic pulmonary affections. Hesse[6] and Heim[7] have found the drug useful as a hypnotic in a great variety of chronic diseases. Bloch[8] has found dionin particularly efficacious in the painful affections of women. It may be given in such affections in the form of suppositories. Fromme[9] and Heinrich[10] have found dionin useful in morphinomania. They claim that it appeases the hunger for morphin without risk of habituation. Nicolaier,[11] Darier,[12] and Graefe[13] have found dionin useful as a vascular stimulant in chronic inflammatory affections of the cornea. It is used as a powder or in 10% aqueous solution. [The difficulty with all these preparations is the difficulty with morphin itself—the formation of a habit. Until it is proved that they can not induce a habit, they must be used with caution. It is not enough to state that "thus far no instance of addiction has been reported."]

**Dormiol.**—Dormiol is the trade name of amylene-chloral. This compound appears as an oily liquid having a camphor-like odor and a cooling taste. It is insoluble in water. Meltzer[14] states that he has used during the last year 20 ounces of dormiol as a hypnotic, and was successful with it in 75% of male and 80% of female patients. He regards it as a very good hypnotic, similar to chloral hydrate, which promptly produces sleep when given in doses of from 7½ to 45 grains, without unpleasant after-effects, even in insane patients, who are in a more or less excited mental condition. He prescribes it with syrup and mucilage. Schultze[15] states that he has administered dormiol 1000 times in the asylum at Andernach. Sleep usually appeared within an hour of its administration. The average duration of its action was from 5 to

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[1] Therap. Monatsh., Sept., 1899.

[2] Deut. med. Woch., No. 44, 1899.

[3] Münch. med. Woch., No. 51, 1899.

[4] Presse méd., No. 43, 1899.

[5] Merck's Arch., June, 1900.

[6] Wien. med. Bl., No. 22, 1899.

[7] Klin -therap. Woch., No. 46, 1899.

[8] Münch. med. Woch., No. 51, 1899.

[9] Berl. klin. Woch., No. 14, 1899.

[10] Wien. med. Bl., No. 11, 1899.

[11] Woch. f. Therap. u. Hyg. des Aug., No. 4, 1899.

[12] Clin. Ophthal., No. 33, 1899.

[13] Therap. Beil. d. Deutsch. med. Woch., XXXVI, p. 9.

[14] Deut. med. Woch., No. 18, 1899.

[15] Neurol. Centralbl., No. 6, 1900.

8 hours. The drug was effective in about 75% of the cases. The best results were obtained in melancholia and hypochondria. The author is of the opinion that dormiol about equals trional in efficiency, but that the one often succeeds where the other fails.

**Epicarin.**—This is described as a condensation product of cresotinic acid and naphthol. It is a reddish powder, acid in reaction, and freely soluble in water and alcohol. According to Frick and Müller, it is a useful remedy in scabies. According to Kaposi,[1] the action of epicarin resembled that of beta-naphthol. Pfeiffenberger [2] found it useful in scabies. It is best used in the form of a 10% ointment.

**Ergot in Asthma.**—Bell [3] speaks highly of ergot and its derivatives in cases of spasmodic asthma. He claims that the drug seldom fails to cut short the paroxysm, and argues from this that we have to deal with a pulmonary vasodilation.

**Erythrol Tetranitrate.**—Walsham [4] believes that in erythrol tetranitrate we have a valuable vasodilator. He has used it with good effect in 3 cases of aortic regurgitation accompanied by cardiac pain. In 2 other cases amyl nitrate was more effective. Of 3 cases of chronic interstitial nephritis 2 were benefited. In one case of Raynaud's disease the effect was very happy.

**Ethyl Bromid.**—Kempter [5] pleads for a more extensive use of this drug as an anesthetic in minor surgery. He quotes German statistics to show that of 60,000 patients anesthetized with bromid of ethyl there were but 16 deaths. Its main advantage is rapid anesthesia (30 to 60 seconds). Nausea and vomiting are rare, and there are no unpleasant after-effects. A fresh preparation is absolutely requisite. From 1 to 3 drams, according to age, are poured in a cone, and the latter is not removed until anesthesia is produced. As in the administration of ether, no air is admitted. Anesthesia lasts from 1 to 2 minutes. Fowler [6] reports exceedingly favorable results in upward of 100 cases from the use of ethyl bromid preliminary to the use of ether. When anesthesia has been induced, he begins with the ether without changing the inhaler. Krusen [7] believes that ethyl bromid is the ideal anesthetic for use in obstetric and gynecologic practice.

**Ethyl Chlorid.**—Lotheissen [8] states that statistics show that ethyl chlorid stands, as regards its mortality, next to chloroform—the latter giving 1 death to 2075; ethyl chlorid, 1 death to 2550. It produces anesthesia quickly, without discomfort, and without unpleasant after-effects. The author reports a case of death from asphyxia under ethyl-chlorid anesthesia in a man with fatty heart and sclerosis of the vessels.

**Eucain.**—Poole [9] concludes as follows from his use of this drug in a series of cases: (1) Eucain is decidedly less toxic than cocain, therefore superior to it; (2) its aqueous solutions keep well and can be sterilized by boiling without destroying the activity of the drug; (3) it produces anesthesia as well as, and sometimes better than, cocain; (4) it is superior to cocain, in that it causes no heart depression or other unpleasant systemic effect; (5) it causes no mydriasis and no disturbance of accommodation, which is an advantage in some cases; (6) it is less dangerous to the cornea than cocain, inasmuch as it does not cause a desquamation of the superficial epithelium.

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[1] Merck's Bericht, 1900.

[2] Klin.-therap., VII, p. 586.

[3] Therap. Gaz., Feb. 15, 1900.

[4] Brit. Med. Jour., Nov., 1899.

[5] Maryland Med. Jour., Sept. 2, 1899.

[6] N. Y. Med. Jour., April 28, 1900.

[7] Phila. Med. Jour., Nov. 3, 1900.

[8] Münch. med. Woch., May 1, 1900.

[9] Jour. Am. Med. Assoc., Oct. 14, 1899.

**Eumenol.**—Müller [1] states that this remedy is extractum radicis tangkui, which has been used in China for centuries in the treatment of dysmenorrhea and amenorrhea. He has found it very useful in delayed menstruation and dysmenorrhea. When the discharge is scanty and watery, eumenol makes it more profuse and heavier. The drug is free from toxic properties and does not act as an oxytocic. The dose is a teaspoonful thrice daily.

**Eunatrol.**—Eunatrol, or oleate of sodium, has been recommended by Cipriani [2] in hepatic colic.

**Euphthalamin.**—This is hydrochlorid of the mandelic acid derivative of eucain B. According to Hinshelwood,[3] euphthalamin approaches more nearly to the ideal mydriatic than any other drug. This view is shared by Vinci,[4] Hale,[5] Jackson and Schneideman,[6] and others. Hale's conclusions from the study of the drug are as follows: (1) Euphthalamin (5 to 10% watery solution) produces no subjective symptoms. (2) It causes practically only mydriasis, pronounced, but of short duration, which shows itself, as a rule, in 30 minutes; earlier, therefore, than in other drugs. (3) Its effect shows itself earlier in youth than in old age, but the difference is negligible. (4) It has no effect on ocular tension; at least, no contrary reports have as yet appeared. (5) It causes no hyperemia or ischemia of the conjunctiva, and it has no effect on corneal epithelium. (6) It has no effect on accommodation, at least in those cases in which the general practitioner would most probably use it. (7) The normal condition of the pupil is rapidly restored. (8) It is apparently nonpoisonous. Jackson and Schneideman remark regarding the risk of producing an outbreak of glaucoma with euphthalamin, that, while it is probably less than with any other mydriatic but cocain, and most writers speak of it as *nil*, we must remember that every other new mydriatic has been put forward with the same assertion, but experience has in each case failed to substantiate the claim. Wood [7] believes that we have in euphthalamin a mydriatic pure and simple—one that is more rapid in its action than cocain and not so lasting as homatropin. He has found that the time of its action can be lessened by combining it with cocain. He recommends the following combination: Euphthalamin and cocain muriate, of each, 0.5% in distilled water. Two drops to be instilled every 5 minutes for a quarter of an hour.

**Europhen.**—Saalfeld,[8] after 7½ years' experience with this remedy in the treatment of ulcerations of the genitals, concludes that, on account of its lightness, odorlessness, and freedom from irritating and toxic properties, it is to be preferred to aviol, orthoform, and iodoform. Flick[1] has found europhen very useful in phthisis. He directs that from 1 to 4 teaspoonfuls of the following ointment should be rubbed into the armpits and inside of the thighs once or twice a day: Europhen, 1 dram; oil of rose, 1 drop; oil of anise, 1 dram; oil of olives, 2½ ounces.

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[1] Münch. med. Woch., June 13, 1899.

[2] Deut. med. Zeit., No. 57, 1899.

[3] Brit. Med. Jour., Sept. 23, 1899.

[4] Therapist, Dec. 15, 1899.

[5] Chicago Med. Recorder, Feb., 1900.

[6] Am. Jour. Med. Sci., June, 1900.

[7] Oph. Rec., April, 1900.

[8] Therap. Monatsh., XIV, No. 3, 1900.

**Fersan.**—According to Silberstein,[2] fersan is a ferruginous preparation made by the action of pure hydrochloric acid on red blood-cells. It appears as a brown powder with a slightly salty taste, and it does not coagulate on heating. As it is not affected by artificial gastric juice, it is surmised that it is absorbed only from the intestine, and the fact that the stools are not bleached as by other iron salts is offered as proof of its complete absorption. Pollak,[3] who used it in 50 phthisical patients, found that it produced a speedy increase in the hemoglobin and of the body-weight. He gave a coffeespoonful in a tumbler of milk and water half an hour before meals.

**Formaldehyd.**—The value of this agent as a disinfectant has now been thoroughly established. According to Park, it is the best disinfectant at present known for the surface disinfection of infected dwellings. For heavy goods it is far inferior in penetrative power to steam and dry heat at 230° F., but for the disinfection of wearing apparel, furs, leather, upholstery, books, and the like, which are injured by great heat, it is, when properly employed, better adapted than any other disinfectant known. Crawford[4] believes that formalin has not received the consideration at the hands of obstetricians and surgeons which it merits. It is less irritating to the hands and wounds than any other efficient drug. It does not injure instruments and is practically innocuous. He recommends a ½% solution for the hands and surface of the body, and ¼% or ⅛% solution for cavities. One dram of formalin added to 3 pints of water makes a ¼% solution. Inhalations of formalin vapor find favor with many practitioners in the treatment of phthisis. Thomas[5] recommends a 4% aqueous solution of the commercial preparation (40% formaldehyd), to be used in a nebulizer. He asserts that patients acquire an increasing tolerance to formalin vapor, and by degrees the strength of the solution can be raised to 20%. Green[6] has found the following mixture most satisfactory for inhalation: Formalin, 1 dram; glycerin, 4 drams; water, 5 ounces. To be used from 4 to 6 times daily. Harrington[7] states that formaldehyd fumes will completely check the vomiting and control the paroxysms of cough. A sufficient amount of gas for an ordinary room is generated by placing 2 or 3 paraform tablets in half a dram of alcohol, and allowing them to evaporate over a gentle heat. If the fumes become irritant, they will do more harm than good.

[1] Penna. Med. Jour., Oct., 1899.

[2] Therap. Monatsh., July, 1900.

[3] Wien. klin. Woch., June 21, 1900.

[4] N. Y. Med. Jour., June 30, 1900.

[5] Western Med. Rev., No. 10, 1899.

[6] N. Y. Lancet, vol. XXI, No. 4.

[7] Ann. Gyn. and Ped., No. 10, 1899.

In very severe cases he also recommends the administration of bromoform. Herschfeld[1] confirms the value which many observers have placed upon formalin in the treatment of hyperhidrosis. He recommends equal parts of formalin and absolute alcohol, to be applied to the sweating parts on alternate days. Adler[2] also prefers formalin to all other remedies in the treatment of hyperhidrosis. When rhagades exist between the toes, he heals them with tannaform before using the formalin. Daniel[3] speaks favorably of applications of undiluted formalin for the removal of warts and other epithelial hypertrophies.

**Gelatin.**—Since Carnot[4] first drew attention to the hemostatic properties of gelatin the drug has attracted considerable attention. Schwabe[5] reports a case of hematuria in a patient with recurrent nephritis in which he employed without success the usual hemostatic remedies. Prompt and permanent relief, however, was obtained by the injection into each infraclavicular region of 7 drams of physiologic salt solution containing 2% of pure gelatin, followed by the daily internal administration for a week of a pint of 10% gelatin solution. According to the author, the pain was not intense and no unfavorable after-effects were observed. On the other hand, Freudweiler[6] reports 2 cases of hemorrhagic nephritis in which the use of gelatin brought on a marked hemoglobinuria, and increased the amount of albumin and the number of tube-casts. In another case uremia quickly developed after the use of gelatin. Arcangeli[7] has employed a normal saline solution, with gelatin in the proportion of 2%, carefully sterilized, in 2 cases of purpura with recurrent epistaxis and bleeding from the gums in young girls, aged respectively 13 and 10 years. In the first patient 2 injections of 20 cc. each, injected with an interval of 24 hours into the subcutaneous tissue of the abdomen, sufficed to arrest completely the hemorrhage from the nose and gums. In the second case the same result was obtained with injections of 15 cc. and 10 cc. Nichols[8] reports a case of profuse hemorrhage from a wound of the arm, which resisted all forms of treatment, but which was promptly arrested by pouring gelatin from an ordinary culture-tube into the wound. Gutmann[9] reports a favorable result in a case of melena neonatorum from the use of gelatin administered both by the mouth and rectum. Later reports concerning the use of injections of gelatin in aneurysm have not been so favorable. Fraenkel[10] obtained a good result in one case of aortic aneurysm. Conner[11] reports 3 cases of thoracic aneurysm treated with subcutaneous injections of gelatin. He found the treatment very painful and not particularly beneficial. Futcher[12] reviews the literature of the gelatin treatment of aneurysm, and reports 9 cases in which it was tried in the Johns Hopkins Hospital. Two hundred and fifty cc. of a 1%

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[1] La Semaine méd., XIX, No. 46.
[2] Arch. f. Dermat. u. Syph., Bd. LI, Heft 1.
[3] Deut. med. Woch., No. 49, 1899.
[4] Presse méd., No. 77, 1897.
[5] Therap. Monatsh., June, 1900.
[6] Centralbl. f. innere Med., July 7, 1900.
[7] Treatment, Nov. 9, 1899.
[8] Med. News, Dec. 2, 1899.
[9] Therap. Monatsh., Heft 10, S. 552, 1899.
[10] Klin.-therap. Woch., No. 30, S. 978, 1899.
[11] Phila. Med. Jour., May 12, 1900.
[12] Jour. Am. Med. Assoc., Jan. 27, 1900.

solution of white gelatin in normal salt solution were injected into the subcutaneous tissue of the thigh or abdomen, under strict antiseptic precautions, every fifth day in the later cases. In not a single instance was the aneurysm cured. In 7 of the 9 cases, however, there was appreciable diminution in the subjective symptoms. The author believes it proved that gelatin does materially increase the coagulability of the blood. The injections are frequently very painful. In several instances the injections were followed in from 2 to 4 hours by a chill and a decided rise of temperature. In no case was there local suppuration. Although no case was cured, the author believes that the treatment is not without merit.

**Glutol.**—This is a combination of gelatin and formaldehyd. It appears as a gray, odorless, and tasteless powder. Henry [1] says that the formaldehyd is set free only in the presence of tissues which are still vital, and not by contact with dead tissue. The author recommends it as antiseptic in those cases in which iodoform has up to the present proved of greatest service, the advantages being the liberation of an antiseptic principle more active than iodoform and the absence of odor and of toxic or irritant properties.

**Glycerin.**—Hermann [2] claims that he has obtained good results in 60% of 115 cases of nephrolithiasis in which he employed glycerin, the concretions being discharged or the pain relieved. Rosenfeld, Ortner, Kugler, and Casper have also spoken favorably of glycerin in renal calculus. The dose of the glycerin was from 1 to 4 ounces in an equal quantity of water, between meals, and repeated two or three times in several days. The only unpleasant effects were headache in 12 nervous patients, and diarrhea in 3 patients who had indigestion. Albuminuria is not considered a contraindication. The good effects are not attributed to the solvent action of the glycerin, but to the physical changes which it effects in the urine.

**Guaiacol.**—Guaiacol and its various compounds are still extensively used in the treatment of phthisis, purulent bronchitis, bronchiectasis, and bronchopneumonia. The compound which has attracted the most attention is the potassium salt of guaiacol-sulphonic acid, known as **thiocol,** and which contains 60% of pure guaiacol. It has the advantages over the older preparations of being tasteless, odorless, and freely soluble in water, and therefore more readily absorbed. The usual dose is from 45 to 90 grains a day, but much larger doses can often be given with advantage. Among those who have spoken favorably of thiocol in the treatment of phthisis may be mentioned Schnirer,[3] Moir,[4] Frieser,[5] Smithwick,[6] and Renzie and Boeri.[7]

The glycerol ether of guaiacol, known as **guaiamar,** is made by the action of anhydrous glycerin on pure guaiacol. It is a white crystalline powder, soluble in 20 parts of cold water. It has a bitter, aromatic taste. Butler [8] states that the drug is antiseptic chiefly through the liberation of nascent guaiacol, partly in the stomach, but chiefly in the intestine. He believes that it has a special advantage in that it stimulates appetite and digestion. Whalen[1] recommends guaiacol very highly in malaria when quinin has not been successful. The dose is from 5 to 45 minims, gradually increased unless disturbed digestion results. From experience with 4 patients the author believes it is best to increase the dose to complete toleration. Christian[2] states that he has used guaiacol locally in 60 acute cases of walking epididymitis with most gratifying results. The application of the drug was followed in all cases by a smarting and tingling sensation in the skin lasting for about an hour. Dermatitis did not occur in any case. The method pursued in carrying out this line of treatment was as follows: The testicle was first gently massaged with a small amount of a 20% guaiacol ointment made up with lanolin. Some of this ointment was then spread upon lint and applied over the affected testicle; the whole of the scrotum was enveloped in a layer of absorbent cotton, and over this was applied a snugly fitting laced suspensory bandage. In many cases where this bandage could not be procured the dressing was kept in place by means of a 1½ inch gauze bandage. This dressing was reapplied every other day. At the end of about 6 days, when the inflammation and pain had entirely subsided and the testicle could be easily handled, an ointment of belladonna, mercury, and ichthyol was substituted for the guaiacol. Nuss[3] and Perry[4] also speak well of this plan of treatment. Funk[5] reports 2 cases of disseminated lupus in young children in which applications of guaiacol twice a day produced a cure, in the one case in 2 months, and in the other in 3 months. The treatment is free from pain, and, according to the writer, it is of value only in disseminated lupus.

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[1] Thèse de Lyon, 1899.

[2] Med. Chron., Jan., 1900.

[3] Klin.-therap. Woch., No. 36, 1899.

[4] Therapist, April 16, 1900.

[5] Therap. Monatsh., Dec., 1899.

[6] Merck's Arch., May, 1900.

[7] Deut. med. Woch., Aug. 10, 1899.

[8] N. Y. Med. Jour., Sept. 23, 1899.

**Heroin.**—Heroin, an acetic ester of morphin, has established for itself a definite place as a sedative for the alleviation of cough. Later reports, however, indicate that it is not so free from a depressant effect on the respiration as Dreser would have us suppose. Harnack,[6] Santesson, Winternitz, and Fraenkel[7] believe it to be more toxic in its action than codein. Nearly all observers agree that $\frac{1}{12}$ of a grain is an average dose, and that larger doses than $\frac{1}{6}$ of a grain should not be given. Although Klink[8] has administered as much as $\frac{5}{6}$ of a grain 3 times a day without untoward effects, larger doses than $\frac{1}{3}$ of a grain a day are frequently followed by nausea, headache, dryness of the throat, vertigo, constipation, or numbness of the extremities. Among those who have found heroin useful in allaying the cough of phthisis, whooping-cough, emphysema, bronchitis, and pneumonia may be mentioned Einhorn,[9] Manquat,[10] Freudenthal,[11] Turnauer,[12] Flockinger,[13] McGee,[14]

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[1] Chicago Med. Recorder, No. 1, 1899.

[2] Therap. Gaz., Mar. 15, 1900.

[3] Merck's Arch., Mar., 1900.

[4] Med. Rec., Jan. 7, 1899.

[5] Klin.-therap. Woch., Sept. 10, 1899.

[6] Jour. de méd., No. 6, 1899.

[7] Münch. med. Woch., No. 46, 1899.

[8] Münch. med. Woch., No. 42, 1899.

[9] Phila. Med. Jour., Oct. 28, 1899.

[10] Bull. méd., No. 89, 1899.

[11] Wien. klin. Rundschau, No. 31, 1899.

[12] Wien. med. Presse, No. 12, 1899.

[13] New Orl. M. and S. Jour., May, 1900.

[14] Cleveland Jour. Med., June, 1900.

Lowenthal,[1] Fulton,[2] Manges,[3] and Daly.[4] Manges' paper is the most comprehensive. In order to ascertain the general opinion of the profession regarding the drug this author sent out letters, to which 141 replies were received. Of these, only 4 were outspoken against it; 50 were, in general terms, in favor of it; 82 represented a large experience of a varied kind. There were 340 cases treated. To these Manges adds 75 cases of his own, making a total of 416 cases. The percentage of failures in the list amounted to 21. The best results were secured in acute bronchitis, pneumonia, phthisis, emphysema, and asthma. In the last two diseases heroin, according to the author, is distinctly superior to any other drug thus far used. During the attack it should be used freely, and preferably subcutaneously in the form of the soluble hydrochlorid. In 2 cases it was successfully used in breaking off the morphin habit. The after-effects are less frequent and of milder degree than those from morphin or codein. Alkalies should not be employed in solution with the hydrochlorid. The drug is incompatible with apomorphin.

**Hyoscin.**—Noble,[5] from a long experience with this drug in cases of insanity, has reached the following conclusions: All patients do not behave the same under its administration. In acute or recurrent excitement 2 or 3 of every 5 will be benefited. Old people, particularly in feeble health, are more profoundly affected by even moderate doses. It is just as efficacious by the mouth as hypodermically, although its action is less rapid. If after increasing the dose to $\frac{1}{60}$ of a grain the desired effect is not obtained, it is useless to increase it further. Frequent doses are not required, 2 in 24 hours being usually sufficient. It produces sleep normally by allaying cerebral excitement and morbid motor activity. It may be combined with potassium bromid or chloral. For the initial dose $\frac{1}{100}$ of a grain is sufficient. Williamson[6] writes that hyoscin is the only drug which has proved of real service in paralysis agitans. He recommends larger doses than $\frac{1}{150}$ of a grain, dissolved in chloroform-water. The maximum dose of $\frac{1}{75}$ of a grain 2 or 3 times daily by the mouth can be given for long periods without toxic effects, although it is well to watch the symptoms. This not only diminishes the severity of the tremor, but it renders the patient more comfortable; it diminishes the restlessness, which is such a troublesome symptom. Rendle[7] reports a case of acute chorea in a boy, aged 16, in which bromids, chloral, and arsenic failed to give relief. His condition soon became apparently hopeless. Hyoscin in doses of $\frac{1}{200}$ of a grain was given hypodermically twice daily, and was soon followed by improvement. The dose was increased to $\frac{1}{100}$ of a grain twice daily, and within a week the movements had almost subsided.

**Ichthalbin.**—This substance, which is a combination of ichthyol and albumin, appears as a brownish powder, odorless and nearly tasteless. It has been thoroughly exploited during the past year. Rolly [1] reports 28 cases of subacute and chronic enteritis in which ichthalbin was given in doses up to 2 drams daily for a long time without harmful results. On the other hand, it seems to have lessened intestinal fermentation, stimulated the appetite, and increased the body-weight. Wolfe [2] concludes that ichthalbin, being nonirritating and without odor and taste, should be substituted for ichthyol both for internal and external use.

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[1] Phila. Med. Jour., Sept. 8, 1900.

[2] N. Y. Med. Jour., Dec. 30, 1899.

[3] N. Y. Med. Jour., Jan. 13, 1900.

[4] Boston M. and S. Jour., Feb. 22, 1900.

[5] Yale Med. Jour., No. 8, 1900.

[6] Practitioner, No. 382, 1900.

[7] Indian Med. Rec., Aug. 30, 1899.

**Ichthoform.**—This is a combination of ichthyol and formaldehyd. According to Rabon and Galli-Valerio,[3] it is a useful intestinal antiseptic in daily doses of from 30 to 45 grains. In two instances of intestinal tuberculosis, however, no very definite results were obtained.

**Ichthyol.**—Further studies of this drug add to rather than detract from its reputation. The following statement concerning the use of ichthyol in phthisis during the year ending November 1, 1899, is contained in the Annual Report of the Loomis Sanitarium, at Liberty, N. Y.,[4] April, 1900. We have continued throughout the year to use ichthyol in a number of cases showing sepsis and more or less expectoration. The greatest objection to the use of this drug is that the immense daily doses—averaging from 20 to 30 grains, 3 times a day—preclude its employment among patients whose financial means are limited. Its effect in the way of changing purulent sputum is so certain as to be almost specific, and for that reason, together with the percentages of good results attained, it has been used, so far as possible, in cases advanced beyond the incipient stage. Of 64 cases treated, 17 were in the incipient stage, 39 moderately advanced, 8 far advanced. Of this number, 17 % were cured, 63 % improved, 8 % remained stationary, and 12 % grew worse. Douglass [5] believes that ichthyol is the remedy which gives the greatest relief when used locally in cases of atrophic rhinitis. It is his habit to use the remedy in 3 ways: First, by means of a 10 % to 20 % watery solution, applied in pledgets after the nose has been cleaned and allowed to remain for from 15 to 30 minutes, after which oily sprays are used; second, in severe cases with ulceration applied full strength, and gently rubbed into the atrophied mucous membrane for a period of 4 or 5 minutes; and, third, as a salve,—40 minims of ichthyol, 5 grains of menthol, 1 ounce of petrolatum,—to be introduced into the nostril at home. According to Clarke,[6] ichthyol in solution, 1 : 5 to 100, injected into the urethra, is a remedy superior to all others in the treatment of gonorrhea in women. Schütze [7] and Müller [8] have had excellent results from the use of ichthyol in burns. The former uses it in solutions of 50 % to 80 %; and the latter, pure and liberally sprinkled with talcum powder. Seibert [9] has treated 56 cases of scarlet fever with inunctions of ichthyol (5 % to 10 % ichthyol-lanolin every 6 to 12 hours). From 1 to 3 ounces of ointment were pressed into the skin so as to cause it to come in contact with the bacteria in the capillaries. The objects attained were: subsidence of swelling, relief of itching, prevention of cutaneous abscesses, decrease in temperature, and lessening of restlessness and insomnia. Stephenson [1] has found the addition of from 10 to 30 minims of ichthyol to the ounce of the various mercurial ointments of service in particular cases of blepharitis.

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[1] Merck's Arch., Aug., 1900.

[2] Merck's Arch., Jan., 1900.

[3] Therap. Monatsh., Heft 4, S. 202, 1900.

[4] Merck's Arch., April, 1900.

[5] Post-Graduate, XV, p. 776, 1900.

[6] Am. Jour. Med. Sci., April, 1900.

[7] Aerzt. Rdsch., No. 5, 1899.

[8] Aerzt. Rdsch., No. 21, 1899.

[9] Merck's Arch., May, 1900.

**Iodalbacid.**—This is a combination of iodin (10%) and albumin. Zuelzer,[2] who has made a careful study of the action of this compound, finds that iodalbacid never produces so acute an excess of iodin, liberated in the tissues, as potassium iodid. Iodalbacid is also practically not eliminated by the kidney, and hence during its sojourn in the body not only is its action (in liberating iodin) slower and steadier, but its excretion from the system is slower. Hence its action is also more protracted. In numerous cases of syphilis and psoriasis in which potassium iodid produced symptoms of iodism the administration of iodalbacid was found to be quite free from such effects. Fifty cases of syphilis were treated with iodalbacid with good results. From 3 to 5 grains in capsules could be taken for the purpose.

**Iodinpyrin.**—This is a combination of antipyrin and iodin, containing 40% of the latter. It is without odor and taste. With acids it forms salts. Junkers [3] has employed the hydrochlorate for the last 8 years in a variety of diseases. As iodinpyrin hydrochlorate is insoluble, it is best given in powder. The dose for an adult is 15 grains every 3 or 4 hours; for a child, 2 to 10 grains 3 or 4 times a day. In these doses the drug proved very useful in muscular rheumatism. In acute articular rheumatism it had great advantage over salicylic compounds in not causing any unpleasant by-effects. Pain at the beginning of menstruation was relieved by suppositories containing from 8 to 15 grains of the drug, and employed every 2 or 3 hours. In some cases of chronic rheumatism iodinpyrin gave rise to the usual phenomena of iodism.

**Iodipin.**—This is an addition product of sesame oil and iodin (10% or 25%). According to Winternitz, it is not decomposed in the stomach, but in the intestine. The dose internally is 2 or 3 drams of the 10% solution, while *per rectum* as much as from 5 to 7 ounces may be given in enemas. The 25% iodipin is used for subcutaneous injection, about 10 cc. being injected into the gluteal region or back. The advantages claimed for iodipin over potassium iodid are freedom from gastric irritation and unpleasant symptoms of iodism; the oily taste, however, is sometimes objected to when the drug is given by the mouth. Those who have found iodipin a useful substitute for other forms of iodin in syphilis, asthma, pleurisy, and locomotor ataxia are Frese,[4] Klingmüller,[5] Burkhart,[6] and Rodestock.[7]

**Iron Iodid.**—Wilson [8] recommends the use of iron iodid in certain forms of infective arthritis, especially gonorrheal. The dose was

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[1] Therapist, May 15, 1900.

[2] Brit. Med. Jour., Sept. 2, 1899.

[3] Therap. Monatsh., Nov. 1899.

[4] Münch. med. Woch., No. 7, 1899.

[5] Berl. klin. Woch., No. 25, 1899.

[6] Deut. med. Zeit., 1899.

[7] Therap. Monatsh., Oct., 1899.

[8] Med. News, July 21, 1900.

30 minims 3 or 4 times daily, increased by 1 minim daily until double the original quantity was taken. Under this treatment rapid recovery was effected in 3 cases in which the ordinary remedies had failed.

**Kaolin.**—Wilbert[1] recommends a mixture of glycerin and kaolin for poultices. The formula is as follows: Kaolin, 1000; glycerin, 1000; boric acid, 100; oil of peppermint, 1; oil of wintergreen, 1; oil of eucalyptus, 2. The kaolin is heated to 212° F. for an hour or more in order to sterilize it; the glycerin is added and heat continued for from 30 to 40 minutes. When nearly cool, the remaining ingredients are added and mixed thoroughly. To prevent absorption of moisture it should be preserved in air-tight receptacles. It has the advantages of being readily applied without boiling or other preparation, of not requiring renewal for from 12 to 24 hours, and of giving almost immediate relief from pain in most instances of acute or subacute inflammation. Moreover, it is not greasy, and can be readily washed off with cold water. [It is much more agreeable if a layer of gauze is interposed between the poultice and the skin. It is a very satisfactory local application.]

**Kryofin.**—This is a phenetidin derivative closely allied to phenacetin. Breidhenstein[2] concludes from his study of the drug that it is an excellent and reliable antipyretic and analgesic, which does not possess any unpleasant by-effects, and which is often of value when other well-known remedies have failed.

**Largin.**—This is a silver-albumin compound containing 11.1% of silver. It appears as a gray, granular powder, soluble in water in the proportion of 1 : 10. Its solutions are precipitated neither by albumin nor by chlorids, so that it has considerable penetrative power. Many observers claim that it is the most useful of the silver-albumin compounds in the treatment of gonorrhea. After cleaning the urethra, injections of largin in solution (0.25%, gradually increased to 2%) are made 2 or 3 times daily. Among those who have placed a high value on the use of largin in gonorrhea may be mentioned Porges,[3] Feleki,[4] and Fürst.[5] Almqvist,[6] Welander,[7] and Stephenson[8] have used largin successfully in inflammatory affections of the eye. Stephenson thus summarizes the results of his experiments in 100 cases of eye diseases: In gonococcal ophthalmia largin was distinctly inferior both to protargol and to silver nitrate. On the other hand, in acute contagious ophthalmia, due to the Koch-Weeks bacillus, cure soon followed the daily application of a 10% solution. In this affection largin has two advantages over lunar caustic: it causes little, if any, pain; it never gives rise to a conjunctival eschar. In acute trachoma largin acts admirably. In diplobacillary ophthalmia the zinc salts are preferable. In acute blepharoconjunctivitis, often the result of infection by pus organisms, largin renders good service, applied both as a 5% ointment and a 10% lotion.

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[1] Am. Jour. Pharm., No. 10, 1899.
[2] Therap. Monatsh., Mar., 1900.
[3] Wien. med. Presse, No. 44, 1899.
[4] Wien. klin. Rundschau, No. 47, 1899.
[5] Dermat. Zeit., vol. IX, 1899.
[6] Arch. f. Dermat. u. Syph., L, No. 2, 1899.
[7] Woch. f. Therap. u. Hyg. des Aug., No. 44, 1899.
[8] Therapist, May 15, 1900.

In some cases of lacrimal trouble excellent results were obtained from injections of largin in addition to the classic treatment. Under such circumstances the drug seemed to exert almost a specific action against the products of suppuration. The only drawback to largin is its liability to stain the conjunctiva if applied for more than a few weeks. It seems even more likely to cause the discoloration than either lunar caustic or protargol.

**Levurine, or Dried Extract of Beer Yeast.**—A. R. Simpson,[1] of Edinburgh University, reports some very encouraging results from the administration of levurine, a dried extract of beer yeast, in sepsis. The drug may be taken in beer or aerated water, or, to those for whom the taste is too repugnant, in cachets. The dose is a teaspoonful, to be given before food, 3 times a day if necessary, until the desired effect is attained of arrest of suppuration and reduction of temperature. In 3 cases of puerperal sepsis the action of the drug was very favorable. Bruce, who tried levurine in a case of carbuncle, writes: "The case of a fairly large carbuncle, which had been increasing until the levurine was begun, seemed to have in 3 days almost entirely gone by absorption without material sloughing; and in 2 days more the surrounding hardness with it. In a similar case I should certainly try it again." Whether the levurine produces its effect by counteracting, as Cassaët and Beylot suggest, a latent glycemia, or by acting as a germicide to pyogenetic organisms, or as an antagonist to their toxins, Simpson does not attempt to decide, but the results of its administration in the cases reported were so striking as to justify him in recording them.

**Liquid Air.**—White [2] has obtained excellent results from liquid air as a local anesthetic, the only precaution necessary being freezing the part solid. The application causes no pain, except slight tingling. Varicose, specific, and chancroidal ulcers treated with spray were promptly healed, one or two applications a week sufficing. Boils, carbuncles, and buboes, in the early stages, were aborted when the parts were thoroughly frozen. Spraying with liquid air gave speedy relief in sciatica and in intercostal and trifacial neuralgia. Liquid air is not antiseptic, but the great cold inhibits bacterial activity, and repeated applications thus serve the purposes of antisepsis.

**Magnesium Chlorate.**—Herscher and Gaucher [3] have used this drug successfully in the form of a 20% ointment for the removal of epithelioma of the lips.

**Mammary Gland.**—Shober [4] states that the desiccated gland of the sheep in daily doses of from 1 to 2 drams has no unpleasant systemic effects, but seems to act on the uterus, causing contraction, diminishing the blood supply, and controlling bleeding. He believes that the remedy is more reliable than ergot. In uterine fibroids with menorrhagia bleeding can be brought under control in a few weeks, the health improves, and the tumors diminish in size up to a certain point. The author has also used the gland with success in many cases of subinvolution.

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[1] Scottish M. and S. Jour., April, 1900.

[2] Med. Rec., July 22, 1899.

[3] La Semaine méd., No. 49, 1899.

[4] Jour. Am. Med. Assoc., July 28, 1900.

**Mercuriol.**—This is an amalgam of mercury, aluminium, and magnesium, containing 40% of mercury in a finely divided state. In the presence of air and moisture magnesium and aluminium oxids are formed, while mercury is set free in the form of minute globules. It has been especially recommended for use on pads in the treatment of syphilis after the method of Welander.[1] Ahman [2] reports 30 cases of syphilis treated with mercuriol. Seventy-five grains were used daily for a period of 5 or 10 days, and subsequently every other day. The remedy was placed in a woolen bag, which was worn next to the skin. Mercury was recovered from the urine in every instance. The duration of treatment ranged from 30 to 40 days. The author regards this method of treatment as more effective and cleanly than ordinary inunction. Hall [3] has also obtained satisfactory results with this method of treatment.

**Mercurol.**—This is a compound of mercury with yeast nuclei, and was introduced by Schwickerath.[4] It contains about 10% of mercury. Fraley [5] reports 14 cases of gonorrhea which were treated with injections of mercurol (1%). Of these cases, 6 were cured in less than 4 weeks, 3 practically cured in 3 weeks, 3 distinctly improved in 16 days, and 2 not improved, although temporarily benefited. In those cases which involved the posterior urethra the results were not so good. Valentine [6] reports a single case of gonorrhea treated with mercurol (5%) in which the gonococci disappeared definitely on the second day. In other cases the results were not so satisfactory. Guiteras [7] reports 150 cases of gonorrhea treated with mercurol. The average strength best borne was 2%. Complications resulted in only 2 cases—1 of gonorrheal rheumatism, 1 of epididymitis—and posterior urethritis resulted in only 1 case. Guiteras believes that the drug destroys the gonococci. When the latter are no longer present, it is better to change to mild astringent solutions.

**Mercuric Chlorid.**—Ingalls and Yeager [8] report 36 cases of smallpox treated with corrosive sublimate baths without mortality. A 6-foot tub was placed beside the patient's cot and filled with a warm solution of bichlorid, 1 : 10,000, and the patient placed therein, head and shoulders above the solution, for 10 or 12 minutes twice daily. The authors conclude that the baths mitigate all the unpleasant features of the disease. Baccelli [9] has obtained excellent results from intravenous injections of corrosive sublimate on animals inoculated with bubonic plague.

**Mercury Biniodid.**—Burgess[10] concludes from a comparative study of the various disinfectants—biniodid of mercury, corrosive sublimate, lysol, carbolic acid, chlorinated lime, and formalin—that biniodid of mercury is the most efficacious, being especially better than the bichlorid because it does not form an albuminate of mercury in the presence of albumin.

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[1] Arch. f. Dermat. u. Syph., vol. XLVI, 1898.

[2] Arch. f. Dermat. u. Syph., April, 1899.

[3] Therap. Gaz., May 15, 1900.

[4] N. Y. Med. Jour., Sept. 30, 1899.

[5] Therap. Gaz., Nov. 15, 1899.

[6] Phila. Med. Jour., May 19, 1900.

[7] Lancet, Sept. 22, 1900.

[8] Merck's Arch., May, 1900.

[9] Lancet, Dec. 16, 1899.

[10] Lancet, June 23, 1900.

**Mercury Tannate.**—Cooper [1] prefers this salt to other preparations of mercury for internal use in the treatment of syphilis because it is not acted upon by the gastric juice, but passes unchanged into the duodenum, where it is decomposed by the alkaline secretions. The author claims for it the following advantages: It does not derange digestion so much as preparations of mercury; it is quickly absorbed and eliminated; it does not produce stomatitis so quickly as other preparations, since it has no cumulative properties.

**Methylene-blue.**—Czyhlarz and Donath [2] conclude, from experiments on the elimination of methylene-blue through the kidney in health and in disease, that there is a retardation of the elimination of the drug through the urine in nephritis. The authors, however, were unable to discover any difference between the various forms of nephritis in this respect. Retardation of elimination is not peculiar to nephritis. The "Lancet" of December 30, 1899, gives the following summary of the uses of methylene-blue: Methylene-blue administered in the form of pill, with about half its weight of powdered nutmeg, and in doses of from 1 to 6 grains 3 times a day, owing to its recognized selective affinity for the nerve-cells and axis-cylinders in living animals, has proved of considerable value in such affections as sciatica, migraine, neuralgia, and herpes. In diabetes mellitus methylene-blue appears to act somewhat in the same manner as antipyrin. When given in full doses up to 6 grains 3 times a day during a period of 6 weeks, sugar has been found to disappear from the urine. Locally the drug diminishes the pain of cystitis and gonorrhea, the urine being highly charged with the drug and acquiring a bright green or blue color when it is given internally. In cases of gonorrhea complicated with rheumatism or neuritis it has proved of special advantage. In simple rheumatism the drug has also been given with good effect, as it has in intermittent fever, both in the acute stage and for the relief of the after-effects, such as enlargement of the liver and spleen. The general effects of the drug resemble those of antipyrin and salicylic acid; when given after food with powdered nutmeg, it excites little irritation in the stomach, the most noticeable feature at the time being the coloration of the urine, of which patients should be warned. Klemperer [3] presents notes on 27 cases of sciatica in which methylene-blue was employed. In 8 of these no effect was produced; in 6 it gave remarkable results; and in 13 the patients were considerably benefited. According to the author, the painful urination occasionally excited by the drug is prevented by an equal amount of powdered nutmeg given with each dose. Bodoin [4] extols the action of this drug as a sedative in various forms of insanity associated with great mental excitement. In 14 cases, including the different types of mania, paranoia with delirium, chronic alcoholism, and hystero-epilepsy, the remedy proved very satisfactory. When injected into the gluteal muscles, in doses of 1 to 1½ grains, it exerted a quieting influence within a few hours, which lasted from 1 to 4 days. No untoward effects were observed. Smithwick [1] reports a series of 50 cases of different types of malaria, in all of which he administered methylene-blue with good results. The usual dose was 2 grains, 6 times daily, in pills or capsules. His conclusions are as follows: Methylene-blue is a perfect succedaneum for quinin. No bad effects follow its use when it is given intelligently. It is the remedy to use in malaria with hematuria, as it acts in a twofold manner. It is the remedy to be given in malaria occurring during pregnancy, as it has no oxytocic effect and as it causes a freer action of the kidneys. Ollwig [2] also believes that methylene-blue is of distinct value in the treatment of malaria, but he considers it far inferior to quinin in the prevention of relapses. O'Neill [3] has found methylene-blue useful in the treatment of gonorrhea, and states that gastric disturbance was not seen when the drug was given with oil of nutmeg and oil of sandalwood. The dose is 1 grain, 3 or 4 times a day.

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[1] West London Med. Jour., April, 1900.

[2] Wein. klin. Woch., June 15, 1899.

[3] Die Therap. d. Gegenw., I, No. 11, 1899.

[4] Klin.-therap. Woch., Nov. 21, 1899.

**Methyl Salicylate.**—Bettman [4] has found local applications of methyl salicylate very efficient in the treatment of gonorrheal epididymitis. From 1 to 2 drams of a mixture of 1 part of methyl salicylate to 2 of olive oil are poured over a piece of nonabsorbent cotton and this is applied to the scrotum. This dressing is covered by a sheet of rubber and the scrotum is supported by a padded suspensory. Within 3 or 4 days the application may be stopped and the usual pressure bandage applied. Caziot [5] also warmly recommends this method of treatment.

**Nirvanin.**—Bolognesi [6] states that this variety of iodoform occurs in the form of a white powder, odorless and having a slightly bitter taste. It is soluble in water and slightly so in ether. It has been employed successfully as a local anesthetic in a great variety of minor operations. A 10% solution produces anesthesia at the point of injection within 5 minutes. The toxic dose is said to be about 165 grains, but according to Braquehayl, not more than ¾ of a grain should be used in operative work. It should be used in stronger solution and in larger doses than cocain, since its anesthetic action and toxicity are less marked.

**Nitroglycerin.**—Wade [7] recommends small doses ($\frac{1}{200}$ of a grain) of nitroglycerin in abdominal palpitations. He believes that the drug does good by lowering the tension in the splanchnic area.

**Orexin.**—Yonge [8] prefers the tannate to other salts of orexin. The tannate appears as a whitish, odorless, almost tasteless powder, insoluble in water, but soluble in dilute acids, and therefore in the gastric juice. According to the author, it shortens the process of digestion by stimulating the secretion of hydrochloric acid and by increasing the motor functions of the stomach. The dose is 10 grains thrice daily before meals, preferably in hot liquid. Zeltner[1] reports 53 cases of anorexia in which he used orexin tannate. Of these, 30 were distinctly benefited, 9 imperfectly, and 14 not at all. The indication for its use is deficient secretion of hydrochloric acid. He recommends it particularly in incipient phthisis, anemia, mild digestive disturbances, and nervous dyspepsia. Bernheim[2] teaches that orexin is contraindicated in cases of hyperacidity, and in cancer and ulcer, because it increases gastric peristalsis.

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[1] Merck's Arch., Feb., 1900.

[2] Zeit. f. Hyg. u. Infektionskrankh., XXXI, No. 2, 1899.

[3] Med. Rec., Mar. 24, 1900.

[4] Merck's Arch., Aug., 1899.

[5] Münch. med. Woch., XLVI, No. 38, 1899.

[6] Bull. gén. therap. méd., Dec., 1899.

[7] Brit. Med. Jour., No. 2007, 1899.

[8] Therapist, No. 5, 1899.

**Orthoform.**—Manquat[3] and Luxenburger[4] have found orthoform particularly useful in ulcerations of the mouth, pharynx, and larynx, and as an analgesic in dysphagia due to cancerous ulceration of the epiglottis or esophagus. According to Manquat, 2½ grains of orthoform in cachet will ease the pain of gastric ulcer in 5 minutes. The drug is also reliable in intertrigo, cracks of nipple, hemorrhoids, and stomatitis. Yonge,[5] Rollet,[6] Brocq,[7] Asam,[8] and Wunderlich[9] call attention to certain untoward effects of orthoform. Asam has encountered sloughing in 9 cases of ulceration in which orthoform had been used. Yonge has observed a similar effect in 2 instances. He infers from a series of 50 cases that the incidence of this complication may be looked for in 4% of all cases. Brocq and other observers have pointed out that orthoform may excite redness, irritation, and severe dermatitis. Epstein has observed vomiting, collapse, and other unfavorable phenomena after the administration of the drug by the mouth. Marsault[10] calls attention to the incompatibility of orthoform with antipyrin. When triturated in a mortar, the two drugs produce a semiliquid mass. Orthoform is also incompatible with silver nitrate.

**Osmic Acid.**—Bennett[11] reports 10 cases of neuralgia treated by injections of osmic acid. The nerve, having been exposed by as small an incision as possible, is hooked up for purposes of fixation. The solution of osmic acid (1.5%), freshly prepared, is injected by means of a sterilized syringe, the needle of which is passed along in the substance of the nerve as far as it will go. The total amount injected should be from 5 to 10 minims, and it should be introduced in 2 or 3 separate injections in order that the whole nerve may be, as much as possible, soaked in the solution. The author claims that the pain promptly disappears in nearly every instance. The tissues are blackened by the acid, but no hindrance to primary union follows.

**Oxygen.**—Wagner[12] believes that the best remedy in hemophilia is oxygen, either by inhalation or by contact. It acts, he claims, in two ways—by causing greater rapidity in the coagulation of the blood, and by causing the nuclei of the endothelial cells of the capillary wall to swell and to narrow the lumen of the vessel.

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[1] Therap. d. Gegenw., I, No. 11, 1899.

[2] Merck's Arch., Jan., 1900.

[3] Arch. de Med., April, 1900.

[4] Die med. Woch., July 10, 1900.

[5] Brit. Med. Jour., July 1, 1899.

[6] Lyon méd., No. 30, 1899.

[7] Presse Méd., No. 30, 1899.

[8] Münch. med. Woch., No. 12, 1899.

[9] Münch. med. Woch., No. 40, 1899.

[10] Jour. de Praticiens, Oct. 28, 1899.

[11] Lancet, No. 3975, 1899.

[12] Physician and Surgeon, Sept., 1899.

**Parotid Gland Extract.**—Mallett [1] and Shober [2] have found this remedy useful in ovarian neuralgia and dysmenorrhea. Shober believes that it should not be used when gross structural changes are present. The daily dose of the dried extract is from 6 to 12 grains.

**Petroleum Oil.**—Robinson [3] believes that petroleum oil is of considerable value in the treatment of phthisis. He gives large quantities, and his experiments have shown that practically all ingested may be recovered from the feces. He believes that its action is entirely mechanical; that is to say, it dissolves and carries off the toxins formed in the intestines. It acts as a germicide by excluding moisture from the bacteria, and is an ideal solvent and diluent for many remedies, such as creasote. Ordinarily its administration results in considerable gain in weight. [The fact that practically all the ingested oil can be recovered from the feces disposes very effectually of the claim that any of the preparations in the market are foods.]

**Picric Acid.**—Local applications of picric acid in various inflammatory conditions of the skin are still favorably spoken of by many writers. Hawthorn [4] reports 12 cases of chancre, hard and soft, in which excellent results were obtained from local applications of picric acid. He first washes the sore with a solution of carbolic acid and then applies a moist dressing of a saturated solution of picric acid. The healthy skin must be protected or otherwise an eczematous inflammation is excited. Delebecque [5] recommends an aqueous solution of picric acid (12 : 1000) in herpes zoster. Absorbent cotton compresses are soaked in this solution, wrung out until partly dry, applied to the affected areas, and covered with dry cotton and a bandage. To avoid maceration of the skin the dressings should be permeable. The applications should be renewed in 3 or 4 days, and removed with care. An alcoholic solution (1 : 10) or an ethereal solution (1 : 20) is more painful when first applied, but is preferred by Thiery. Picric acid acts as an antiseptic and analgesic. It speedily relieves the neuralgic pains and allays the intense itching.

**Pilocarpin.**—Popham [6] has employed pilocarpin successfully in several cases of biliary colic. The drug was given hypodermically in ascending doses. The results were undoubtedly encouraging, and only once did the drug give rise to any unpleasant symptoms. The author regards pilocarpin as more efficient than morphin in hepatic colic, and attributes the good results to the increase of secretions, whereby the passages are lubricated and the expulsion of the stone facilitated. Smith [7] reports good results in chronic otitis media with pilocarpin. He begins with $\frac{1}{16}$ of a grain and gradually increases until he secures profuse sweating. The dose is repeated every alternate day until 6 or 8 doses have been given. Strychnin or whisky may be given with pilocarpin to prevent any untoward effect of the drug upon the heart.

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[1] Deut. med. Zeit., No. 82, 1899.

[2] Phila. Med. Jour., Aug. 4, 1900.

[3] Med. News, July 14, 1900.

[4] La Semaine méd., No. 13, 1900.

[5] Rev. de Therap., No. 20, 1899.

[6] Brit. Med. Jour., June 30, 1900.

[7] Merck's Arch., Mar., 1900.

**Piperazin.**—This remedy has received comparatively little attention during the past year. Fearnley [1] has found it useful in gout, and Aldrich [2] reports 2 cases of renal calculus in which piperazin was used with benefit. Blumenthal [3] states that favorable results have been obtained in Leyden's clinic with piperazin quinate in various gouty disorders. The daily administration of from 75 to 120 grains caused a decrease of from 40% to 50% in the amount of uric acid in the urine, hippuric acid appearing instead. Ewald, Goldscheider, Fraenkel, and Meyer also report favorable results in gout and nephrolithiasis from the use of piperazin quinate.

**Ovarian Extract.**—Mosse,[4] in his monograph on opotherapy, concludes that ovarian extract is most useful in relieving preclimacteric disturbances, although it is also indicated in the reflex troubles following the establishment of the menopause. Menstruation that has been prematurely arrested reappears after the use of the drug, but it has no influence in exciting the menstrual flow in young girls who have the nisus without any discharge of blood. In discussing so-called indirect opotherapy, he states that in cases of amenorrhea iron is preferable to ovarian preparations, while he is confident that they have no value in the treatment of osteomalacia as compared with castration. Arcangell [5] believes that ovarian extract is harmful in chlorosis. Delaunay [6] and Moreau [7] recommend the drug in exophthalmic goiter, especially when the disease is associated with menstrual irregularities, and Marchand [8] has found it useful in epilepsy connected with menstrual disorders.

**Potassium Bicarbonate.**—Luff [9] finds that the gelatinous form of sodium biurate is about 5 times as soluble in blood-serum as the crystalline variety; and that the higher the alkalinity of the blood from the presence of *sodium bicarbonate*, the more rapid and complete the conversion of the soluble gelatinous biurate into the comparatively insoluble and crystalline form. On the other hand, he finds that the greater the proportion of *potassium bicarbonate* present in the serum, the greater is the inhibitory effect on the conversion of the gelatinous biurate into the crystalline form. This inhibitory influence exerted by the presence of potassium bicarbonate explains the well-known beneficial effect of the alkaline potassium salts in the treatment of acute and subacute gout.

**Protargol.**—This remedy has attracted more attention during the past year than any other organic compound of silver. It is a proteid compound containing 8.3% of metallic silver. It is readily soluble in water and its solutions are stable. Emmert[10] publishes a report of 350 cases of inflammatory eye disease in which he employed protargol with marked success. In simple conjunctivitis he used it in solutions containing from 4 to 12 grains to the ounce. He finds that protargol, while containing a high percentage of silver, has the advantage of being much less irritating than many other silver salts, and causes no eschar.

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[1] N. Y. Lancet, XXI, No. 3, 1900.

[2] N. Y. Med. Jour., Sept. 29, 1900.

[3] Bull. Gén. de Thérap., May 15, 1900.

[4] Am. Jour. Med., No. 91, 1900.

[5] Riforma Med., No. 91, 1899.

[6] Presse méd., No. 6, 1899.

[7] Presse méd., No. 5, 1899.

[8] Presse méd., No. 15, 1899.

[9] Brit. Med. Jour., Oct. 28, 1899.

[10] Lancet, Oct. 14, 1899.

Stephenson[1] has found protargol of most value in gonorrheal ophthalmia. He recommends a 50% solution applied twice daily. Ramsay[2] also finds protargol (5% solution) the most efficient remedy in gonorrheal ophthalmia. Gleason[3] believes that protargol in 5% solution is the most efficient of all the organic salts of silver in the treatment of chronic suppuration of the middle ear. Ahlstrom,[4] Baum,[5] Hawkins,[6] and Bowry[7] report successes with protargol in the treatment of gonorrhea. Ahlstrom reports 100 cases in which he used the drug, and claims to be able to abort the disease when seen within 8 days of its onset. He employs a 2% to 4% solution twice a day for the first 5 days, using from 5 to 10 grains for each injection, which is retained 10 to 15 minutes. During the following 5 days a 1% to 2% solution is used. Ruheman[8] employs protargol internally in cases in which he formerly used silver nitrate—gastric ulcer, cancer, diarrhea, and locomotor ataxia. Its advantages are that it is unirritating and does not, as a rule, produce argyria even when long administered. In a case of tabes, however, slight discoloration of the buccal mucous membrane was produced after 26 drams had been taken. The maximum daily dose is 9¼ grains.

**Pyramidon.**—This drug, which is dimethyl-amido-antipyrin, has been studied by Filehme and Spiro,[9] who find that its action is similar to that of antipyrin, but more powerful. Pauli[10] reports 2 cases in which outward symptoms followed the use of the drug in 5-grain doses. The first developed a painful paresthesia and the second an urticarial eruption.

**Quinin.**—Dock[11] concludes that in tertian or quartan intermittent quinin is to be given during the decline of the paroxysm. The hydrochlorate is recommended, followed by 15 drops of hydrochloric acid. In the estivo-autumnal type from 5 to 10 grains should be given at intervals of 5 hours until the temperature remits; then a daily intermittent dose until apyrexia.

**Salipyrin.**—Bendtner[12] concludes that salipyrin has a favorable influence on uterine hemorrhages if there are no severe anatomic changes present. It has a soothing effect upon menstrual troubles and others accompanying uterine hemorrhage. Salipyrin acts exceedingly well in premenstrual and menstrual psychic conditions of depression, although the action is but temporary. The drug is indicated in menorrhagia (with or without diseased adnexa), if not due to cancerous processes, or large tumors, or hemorrhages following parturition and abortion; in climacteric hemorrhages; in hemorrhages occurring some considerable time after confinement or abortion; in threatening abortion; in dysmenorrhea; and in uterine disturbances accompanied by neuralgic and periodic symptoms, and also in menstrual troubles in which no severe disease of the uterus is present.

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[1] Therapist, May 15, 1900.
[2] Edinb. Med. Jour., Jan., 1900.
[3] Laryngoscope, Mar., 1900.
[4] Therapist, Feb. 15, 1900.
[5] Medicine, No. 7, 1899.
[6] Therapist, June 15, 1900.
[7] Thèse de Paris, 1899.
[8] Therap. Beil. d. Deut. med. Woch., Oct. 5, 1899.
[9] Nouveaux Remedes, No. 3, 1900.
[10] Centralbl. f. d. gesammte Therap., Heft 3, S. 129, 1900.
[11] Jour. Am. Med. Assoc., vol. XXXIII, 1899.
[12] Cor.-Bl. f. schweiz. Aerzte, No. 2, 1900.

**Salol.**—Begg [1] claims to have obtained excellent results in smallpox from the use of salol. The drug almost always prevented the vesicles from maturing into pustules. No dangerous effects from the use of the drug in daily doses of 1 dram, continued for a long period, manifested themselves.

**Sanatogen.**—Rybiczka [2] has made a study of this drug. It is a grayish powder, composed of 95% casein and 5% sodium glycerophosphate. It is easily digested and has been used by the author in a variety of affections. It stimulates the appetite, lessens the insomnia of neurasthenics, and increases bodily weight and, in some instances, the hemoglobin content of the blood.

**Santonin.**—Lydston [3] has obtained better results in epilepsy from the use of santonin than from the use of bromids. He begins with 2 grains and gradually increases the amount to the point of tolerance. As a rule, 15 grains 3 or 4 times daily are well borne.

**Sodium Bromid.**—MacLeod [4] recommends the bromid sleep in acute mania and in the morphin and chloral habit. He gives 2 drams of the drug every 2 hours until an ounce is given the first day. On the second day a similar amount is given. Sleep lasts from 5 to 9 days. The patient, however, can be sufficiently nourished with milk. During this sleep the higher nerve-centers are arrested to an extent that can not be attained in any other way. Recovery is gradual and is complete in about 21 or 24 days. MacLeod has induced this condition 9 times with advantage. One patient died on the seventh day from double pneumonia, which was not thought to have had any connection with the treatment.

**Sodium Cacodylate.**—Of all the new drugs, this one has attracted the most attention during the past year. According to Prokhorow,[5] cacodylic acid is one of those arsenic compounds (organic) which can be introduced into the body in considerable doses without producing toxic symptoms. It is split up in the body with extreme rapidity, and the final products are chiefly excreted by the lungs and kidneys. Cacodylic acid or sodium cacodylate has been successfully used in the treatment of phthisis, various cutaneous diseases (psoriasis, lupus, lichen planus, and epithelioma), anemia, and chorea. The dose varies from 1 to 1½ grains hypodermically and from 3 to 6 grains by the mouth or rectum. Gautier[6] states that subcutaneous injections of sodium cacodylate are painless, and in this latent organic form all objections to arsenic as such disappear. When given in this form a hyperleukocytosis results, the red cells are remarkably increased, oxidation is augmented, and thus the blood and tissues are constantly strengthened and renewed. Frassi [7] has studied the action of sodium cacodylate in chlorosis and tuberculosis.

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[1] Lancet, Jan. 27, 1900.
[2] Wien. klin. Woch., Mar. 1, 1900.
[3] Therap. Gaz., No. 15, 1900.
[4] Brit. Med. Jour., Jan. 20, 1900.
[5] Vratch, No. 19, 1899.
[6] Rev. de Therap., No. 13, 1899.
[7] Gaz. degli Osped., Mar. 18, 1900.

In chlorosis the treatment was always followed by a gain in weight and a marked increase in the elimination of urea. While the hemoglobin was constantly increased, the influence on the number of cells was much more marked. In tuberculosis the general effect of the drug was more noticeable than its local action. Renaut[1] has used sodium cacodylate (¾ of a grain 1 to 5 times a day) successfully in epithelioma of the tongue. Letulle[2] and Sandoz[3] have found the drug a valuable aid in the treatment of tuberculosis. Bormano[4] and Zenner[5] report favorably upon its use in various anemias. Garand[6] reports 3 cases of chorea successfully treated with rectal injections of sodium cacodylate. Gautier, Manchetti, Heirtz, and Rendu object to this administration of the drug by the mouth. They claim that when so administered it imparts an alliaceous odor to the breath, and may become poisonous if it meets with reducing agents in the alimentary canal; but Grasset and Brousse[7] do not subscribe to this view. These observers have used it in many cases by the mouth without observing any intolerance.

**Sodium Eosinate.**—Bourneville and Chapotin[8] report 10 cases of epilepsy in which they used this drug. The dose was 15 grains, gradually increased to 45 grains. Under the larger doses the number of convulsions materially decreased, but after a period ranging between 6 weeks and 2 months serious toxic symptoms appeared which necessitated the discontinuance of the treatment. Poisonous effects consisted in redness and swelling of the face and hands, followed by superficial ulceration, and later by falling-out of the nails.

**Sodium Saccharate.**—Schreking[9] contends that an ideal physiologic saline solution should contain, in addition to calcium and other salts, which have a favorable influence on cardiac action, some substance capable of combining with carbonic acid present in excess in the body-fluids. He finds that sodium saccharate combines with carbon dioxid, and thus neutralizes its toxic effect, the result of the reaction being sugar and sodium carbonate. The solution recommended contains 0.03% of sodium saccharate and 0.07% of sodium chlorid. It is important that the sodium saccharate should be chemically pure, so that the mixture shall contain neither free sugar nor sodium hydrate.

**Sodium Salicylate.**—Brunton[10] says that in persons who are liable to headaches he generally prescribes sodium salicylate, 15, 20, or 30 grains at night, with 10, 20, or 30 grains of potassium bromid. This mixture acts better than either sodium salicylate or potassium bromid alone, and it will usually prevent the occurrence of a headache in the morning. If the headache should come on, he recommends that the dose should be repeated; and in the case of people who suffer from very violent and often recurring headaches, he gives them the salicylate not only morning and night, but 3 times a day in small doses, either immediately before or after meals. For the depression and weakness which the salicylate is liable to produce he associated ½ of a dram of aromatic spirits of ammonia. Although patients may take sodium salicylate for years without apparent harm, it is known theoretically that salicylic compounds have a tendency to produce anemia, and therefore it is advisable in some patients to give with the sodium salicylate, when the latter is used regularly, a little iron.

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[1] Rev. de Therap., No. 7, 1900.

[2] Merck's Arch., Aug., 1900.

[3] Thèse de Paris, 1900.

[4] Gaz. degli Osped. et delle Clin., No. 39, 1900.

[5] N. Y. Med. Jour., LXXI, 1900.

[6] Nouveaux Remedes, XVI, 1900.

[7] La Semaine méd., No. 11, 1900.

[8] Progrès méd., Jan. 6, 1900.

[9] Therap. Monatsh., Dec., 1899.

[10] Brit. Med. Jour., Nov. 4, 1899.

**Sodium Sulphate.**—Buchanan [1] has used sodium sulphate with much satisfaction in 453 cases of acute dysentery. He prescribes the following mixture: Sodium sulphate, 1 ounce; fennel-water, 4 ounces. Of this mixture 4 drams are given 3 or 4 times daily until the stools become bright yellow and show no trace of blood or mucus.

**Sodium Tellurate.**—Barié [2] has employed this remedy for nearly 5 years in a great number of tuberculous cases, and has found it very satisfactory for controlling night-sweats. He has never observed any serious toxic effects (occasionally, when used in unusually large doses, it caused a little colic and diarrhea). The only drawback to its use is the odor of garlic which it imparts to the breath, but this is a matter of little moment. The dose is 4 or 5 grains a day for 4 or 5 consecutive days. In severe cases 2 doses of 4 grains each may be administered. The drug is best administered in pill form.

**Spartein.**—Carrien [3] speaks very favorably of spartein with potassium iodid in cardiopathies with arteriosclerosis. In these cases spartein is often preferable to digitalis, since, unlike the latter, it stimulates the heart without increasing the peripheral resistance. Thomas [4] concludes from experiments on rabbits that spartein slows the pulse and raises the blood pressure. The writer agrees with Cushing and Mathews in attributing the slowing of the heart exclusively to the action of the drug on the cardiac muscle. He believes that the increase of blood pressure is chiefly due to the same cause, but that a vasomotor influence can not be entirely excluded.

**Strontium Bromid.**—Cullinan [5] and Laborde [6] consider this salt preferable to potassium bromid in the treatment of epilepsy.

**Strophanthus.**—Pisani [7] holds that this drug increases the blood pressure solely by its stimulant effect on the heart, and that it has no action on the vasomotor system. Along with the increased pressure there is a reduction in the pulse-rate from 5 to 20 beats a minute. The drug is especially indicated in myocarditis when sufficient fiber remains to respond to the stimulation.

**Strychnin.**—Dana [8] writes, concerning the systemic treatment of tic douloureux by means of heroic doses of strychnin, that after experimenting with this method for over 6 years, in about 15 cases, he has found that in early cases—that is to say, in the first and second years—the strychnin treatment will almost invariably arrest or control the disease in anemic or exhausted subjects.

[1] Brit. Med. Jour., Feb., 1900.

[2] Jour. de Praticiens, Feb. 17, 1900.

[3] La Semaine méd., No. 45, 1899.

[4] Rev. méd. de la Suisse Rom., Dec., 1899.

[5] Lancet, No. 3971, 1899.

[6] Merck's Arch., Feb., 1900.

[7] Gaz. degli Osped. et delle Clin., No. 109, 1899.

[8] Jour. Am. Med. Assoc., May 5, 1900.

**Stypticin.**—Stypticin is the name given to cotarnin hydrochlorid, which appears in the form of yellow crystals, freely soluble in water. The dose is $\frac{1}{2}$ of a grain 3 or 4 times daily. Pradzynski[1] has obtained excellent results with the drug in 30 cases of climacteric hemorrhage and threatened abortion, its action in the latter being particularly good. Mederhoff[2] reports 26 cases in which stypticin was used in the treatment of uterine hemorrhage from various causes. He began with less than a grain, increasing the dose to 6 grains. If administered hypodermically, from $1\frac{1}{2}$ to 2 grains were used daily. No bad effects were noted in any instance. The writer concludes that stypticin has a distinct hemostatic action, especially in metrorrhagia, which is not due to the inciting of uterine contraction. It seems to act rather upon the central nervous system (vasomotor). Gottschalk,[3] Czempin,[4] Schaefer,[5] and Boldt[6] also speak well of the action of stypticin in uterine hemorrhage.

**Suprarenal Extract.**—Reports on the utility of suprarenal extract in the treatment of Addison's disease continue to appear from time to time. Johnston[7] presented a case at the annual meeting (1900) of the Association of American Physicians in which rapid improvement followed the exhibition of 9 grains of the extract 3 times a day. The same author has collected 43 cases of the disease that have been treated with suprarenal extract. Of these, 13 were improved, 9 recovered, 11 died, 3 showed no improvement, and the result was not recorded in 7. Fussac[8] has found suprarenal extract of service as a vascular tonic in neurasthenia, and Stoeltzner[9] has observed good effects from its use in rachitis. Hill[10] has tried a glycerin extract of the fresh gland (8 grains of gland thrice daily) in a large number of cases of epilepsy. Of the results he writes as follows: "I would consider it a success compared with any and all other plans of treatment if there were nothing more in its favor than the striking improvement in the mental and physical condition of the patients." The value of suprarenal extract as a peripheral contractor of the vessels in affections of the eye, throat, and nose has been fully established. Somers[11] draws the following conclusions from the use of suprarenal extract as an astringent in 450 cases: It is nontoxic and nonirritating; it prevents to a marked extent the toxic effects of local anesthetics by retaining them in the tissues and preventing absorption; its activity is not impaired by boiling, and the addition of carbolic acid will preserve the solutions indefinitely without impairing their value; it will prevent primary and greatly lessen the danger of secondary hemorrhage; its action is manifested in 20 seconds, attains its maximum in 5 minutes, and lasts from $1\frac{1}{2}$ to 24 hours; it increases the tonicity of the parts, augments the action of other drugs, especially cocain, and diminishes postoperative swelling. The author highly recommends the following local application in hay-fever: Adrenal, 20 grains; phenic acid, 2 grains; eucain B, 5 grains; distilled water, 2 drams. Macerate 10 minutes; filter. This solution will not decompose nor lose its activity for several months. As regards the power of suprarenal extract to prevent secondary hemorrhage there seems to be some difference of opinion. Hopkins [1] has found after a rather extensive use of the drug in intranasal operations that there is a marked tendency to secondary hemorrhage, and recommends plugging the nose after applications of the extract. A number of specialists to whom he wrote had also experienced the same trouble. Hopkins reports as a more rare sequel to the use of the extract 2 cases of intense coryza.

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[1] Allg. med. Central. Zeit., No. 42, 1899.

[2] Am. Jour. Med. Sci., Dec., 1899.

[3] Therap. d. Gegenw., No. 8, 1899.

[4] Münch. med. Woch., No. 22, 1899.

[5] Deutsche Praxis, Nos. 12–14, 1899.

[6] Med. News, April 8, 1899.

[7] Phila. Med. Jour., May 5, 1900.

[8] Méd. moderne. No. 80, 1899.

[9] Deut. med. Woch., No. 37, 1899.

[10] Bull. of Mount Hope Retreat, 1899.

[11] Merck's Arch., June, 1900.

**Tannalbin.**—This is a compound of tannin and albumin. It is not decomposed by the gastric juice, but by the alkaline secretions of the intestines. The dose is from 5 to 15 grains 3 or 4 times a day. Its action is somewhat slow but persistent. Moore [2] reports a number of cases of diarrhea in which he used tannalbin with advantage. Moncorvo [3] also speaks highly of its action in the diarrhea of children. On the other hand, Friedgring [4] claims that it has no bactericidal powers and does not check the absorption of toxins.

**Tannigen.**—This is an acetic ester of tannic acid. The dose is from 10 to 20 grains 4 or 5 times daily. Clark [5] has found it useful in all forms of diarrhea with watery discharges. It is most suitable for subacute and chronic diarrheas.

**Tannoform.**—This is a compound of tannic acid and formaldehyd. It appears as a rose-colored powder, tasteless, insoluble in water, but freely soluble in alkaline solutions. It is a powerful intestinal antiseptic and astringent. The dose is from 1 to 3 grains, according to age. Goldmann [6] and St. Grosse [7] have found it particularly useful in the acute and chronic intestinal catarrhs of children. Adler [8] has found it more serviceable than formaldehyd in hyperhidrosis of the feet when there is maceration of the parts.

**Terpinol.**—Janowsky [9] recommends terpinol (3 drops at frequent intervals) in hemoptysis.

**Thymus Extract.**—Cohen [10] does not agree with Cunningham and Svĕhla that this drug acts as a vasodilator; on the contrary, he finds it useful in conditions associated with vasomotor ataxia and vascular instability. He records some gratifying recoveries as well as some failures from the administration of the extract in asthma, urticaria, and the neurovascular disturbances of the menopause. Pitres [11] records a case of pseudomuscular hypertrophy benefited by the administration of thymus extract.

**Thyroid Extract.**—Oswald [12] finds that the colloid substance of the thyroid consists of two albuminoids—thyroglobulin and a nucleoproteid. The former is the active component and is the exclusive vehicle of the iodin, and hence the colloid containing it is the only active secretion of the gland. Ewald [1] has confirmed Mabille's observation that arsenic offsets the unpleasant secondary symptoms of thyroid medication. Mahon and Babcock [2] have obtained gratifying results with thyroid treatment in acute mania and melancholia, puerperal and climacteric insanities, and primary dementia. Drewry and Henderson,[3] on the contrary, conclude that thyroid extract is a remedy of very limited, if of any, value in any form of insanity. Porges [4] asserts that thyroid extract is suitable in the treatment of obesity only when there are some traces of a myxedematous tendency—very small or nonpalpable thyroid, doughiness of the skin and subcutaneous tissues, etc. Trouchet,[5] Steinlin,[6] and Murray[7] confirm the favorable reports of Gauthier and others concerning the favorable action of thyroid extract in delayed union of fracture.

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[1] N. Y. Med. Jour., Aug. 25, 1900.

[2] Merck's Arch., Feb., 1900.

[3] Aerzt. Central.-Anz., No. 14, 1899.

[4] Therap. Monatsh., XII, 49.

[5] Therap Gaz., June 15, 1900.

[6] Wien. med. Presse, No. 10, 1899.

[7] Klin.-therap. Woch., No. 17, 1899.

[8] Deut. med. Woch., No. 40, 1898; Therap. Beilage, No. 10.

[9] Klin.-therap. Woch., VII, No. 8, 1900.

[10] Jour. Am. Med. Assoc., Aug. 18, 1900.

[11] Gaz. hebdom. de méd., Jan. 8, 1899.

[12] Zeit. f. physiol. Chem., vol. XXVII, 1899.

**Toxin of Erysipelas (Coley's Fluid).**—Coley [8] publishes a review of the cases of malignant disease treated with his fluid. He limits the use of the fluid absolutely to inoperable cases. Of Coley's own cases, 13 have passed the 3-years limit, one of which had a recurrence later and died 6 months afterward. One remained well seven years, 3 six years, 2 five years, 7 three to five years, 4 two to three years, 3 one to two years, and 3 others less than one year after the completion of the course of treatment. As regards the type of tumor, the most successful cases are those of spindle-celled sarcoma. Of 230 cases treated, in only 2 were serious symptoms induced as a result of the treatment. The initial dose of the fluid should rarely exceed ½ of a minim, and the treatment must be prolonged—6 months, with occasional rests, in some instances.

**Tropacocain.**—Block [9] prefers tropacocain to cocain for use in Schleich's infiltration method of inducing anesthesia. He uses a solution containing 3 grains each of tropacocain hydrochlorate and sodium chlorid in 3 ounces of distilled water. In an extensive experience with this fluid he has never seen the slightest sign of intoxication. This is ascribed to the fact that tropacocain is but one-third as toxic as cocain.

**Urotropin.**—This remedy has lost none of its reputation in the treatment of infective processes in the genito-urinary tract. Thompson[10] summarizes his conclusions concerning the therapeutic action of urotropin as follows: (1) A urinary sterilizer, antiseptic, and acidifier,—prompt and reliable in action, moderate in dose,—which, if adhered to, renders the urine both nontoxic and nonirritating to all parts of the animal economy. (2) In virtue of its peculiar affinity for the urine, into which it passes unchanged, and where it parts with formaldehyd, it is apparent that its action in genito-urinary lesions is likely to be complete and certain. (3) Its decisive and lasting effect, and especially its comparative singleness of action,—which last is a most desirable property,—should give it a place in the list of medicinal specifics. (4) From the observations reported thus far, urotropin has appeared to be most frequently indicated in chronic disease, where it has produced exceptionally good results. (5) In the writer's personal experience the diuretic action of the drug was not marked enough to render it deserving of claim to such a virtue. Dalton [1] recounts 5 cases of posterior urethritis in which most decided benefit resulted from the use of urotropin, after other measures had failed. In 2 other cases doubtful relief was obtained, due, however, the writer believes, in both to the patient's neglect of treatment. The average dose was 7 to 10 grains, 3 times a day, taken well diluted with carbonated water. When the urine is very acid, the writer prescribes alkalies; and when it is very alkaline, mineral acid with gentian. [This remedy has apparently secured a permanent place in therapeutics.]

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[1] Therap. d. Gegenw., No. 9, 1899.

[2] Therap. Gaz., Feb., 1900.

[3] Va. Med. Monthly, v, p. 174.

[4] Prag. med. Woch., xxv, No. 6, 1900.

[5] Jour. de Praticiens, Dec. 9, 1899.

[6] Arch. f. klin. Chir., Bd. LX, Heft 2.

[7] Ann. of. Surg., June, 1900.

[8] Jour. Am. Med. Assoc., April 14, 1900.

[9] Centralbl. f. d. ges. Therap., No. 1, 1900.

[10] Boston M. and S. Jour., Nov. 4, 1899.

**Tuberculin.**—Although the use of tuberculin in the treatment of phthisis has been well-nigh abandoned, a few enthusiastic disciples of Koch still experiment with it, and claim for it distinctly curative properties. Two preparations are employed—"old tuberculin" and "T. R." According to de Schweinitz,[2] old tuberculin is a solution containing a mixture of several foreign substances besides the poisons of the germs, while T. R. is a solution of the substances which have been produced within the cells during the growth of the tubercle bacilli, and in its preparation has never been subjected to a sufficiently high temperature to destroy or injure the germs or their products. Heron [3] states that prior to April, 1891, he treated 37 patients with old tuberculin, 32 having phthisis and 5 having lupus. All the lupus cases improved, but relapsed. Of 32 cases of phthisis, 8 subsequently died; 1, however, remaining well until 1897. Ten of the 32 cases were now fairly well, and 3 hadbeen lost sight of. During the years 1897–1898, 9 cases of phthisis and 1 of lupus were treated with new tuberculin (T. R.). Two of these, hopeless from the start, died. The remaining 7 all improved to a remarkable degree. Heron has not at any time seen a single instance of harm in consequence of treatment with either the old or the new tuberculin. Over 2000 injections have been given in his wards, and in no case had there ever been a superficial abscess at the seat of puncture. The one case of lupus did extremely well; 18 months afterward the scar was still healthy. During the years 1898–1899, 10 cases of phthisis had been treated. In 2 the injections had to be discontinued on account of high fever due to mixed infection. Of the remaining 8 cases, 3 were still in the hospital, and were improving, and 5 came to the hospital weekly and each received an injection of one bottleful of tuberculin. All had improved and were still improving. The remedy, according to Heron, is most suitable in the early stage, and cases with mixed infection should be subjected to other treatment. Petruschky [4] concludes from his work in Koch's Institute that tuberculin is of no value in cases of phthisis attended with secondary infections. He holds that it is best to begin with very small doses,—less than 0.1 of a milligram,—and then to increase rather rapidly. He believes that a permanent cure can be achieved with tuberculin, especially if the patient is subjected to a periodic course of treatment. Usually two courses suffice. The author reports 22 cases, 4 being advanced and 18 milder cases; in all the tuberculin brought about an apparently complete cure. Krause [1] reports 41 cases of phthisis treated with tuberculin. Thirty of these were uncomplicated, while 11 showed mixed infection. He considers that 12 of these were practically cured, temporarily at least; but he admits that in none of these cases were tubercle bacilli found. He states that the earliest injections are likely to cause considerable pain; and when this does not disappear after a few repetitions, he discontinues the use of the tuberculin. He stands almost alone in considering T. R. more dangerous than the old tuberculin.

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[1] Therapist, London, Oct. 16, 1899.

[2] Phila. Med. Jour., Mar. 31, 1900.

[3] Lancet, Oct. 14, 1899.

[4] Berl. klin. Woch., Dec. 11, 1899.

**Validol.**—This is a compound of menthol and valerianic acid. It is a colorless liquid, of cool, slightly bitter taste. The dose is from 10 to 15 drops, on sugar. Goldmann [2] recommends it in neurasthenia; Cipriani,[3] in the treatment of anorexia and vomiting of phthisis; and Vertrun,[4] in the vomiting of pregnancy.

**Vanadium.**—The salts of vanadium—sodium, iron, and lithium—have found favor as alteratives with a number of observers. Anceau [5] has found sodium vanadate a powerful tonic and reconstructive in tuberculosis. He recommends $\frac{1}{32}$ of a grain daily by the mouth. Bertheil [6] has employed vanadium salts in over 140 cases. He prescribes the sodium salt in aqueous solution, in doses of from $\frac{2}{3}$ to $\frac{5}{6}$ of a grain in 24 hours. According to the author, the most important applications of the drug are as follows: In tuberculosis, when good results are at least temporarily obtained. The appetite returns, bodily vigor increases, and the general condition becomes notably improved. Gastric disturbance, however, is noted as an unfavorable feature in these cases. In anemia and chlorosis the results are conclusive, the vanadate of iron being employed. In neurasthenia sodium phosphovanadate has given excellent results. In diabetes the results have been in certain cases very remarkable, especially on the glycosuria and the acetonuria. In gout sodium vanadate, and more especially lithium vanadate, produce marked effects on the subacute articular pain. The vanadium medication, finally, has been known to effect, temporarily at least, the growth of neoplasms, and it has been of service in malaria and in certain skin affections.

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[1] Deut. med. Woch., May 18, 1899.

[2] Klin.-therap. Woch., No. 33. 1899.

[3] Allg. med. Central. Zeit., No. 75.

[4] Berl. klin. Woch., No. 3, 1899.

[5] Thèse de Paris, 1899.

[6] Thèse de Lyon, 1899.

# PHYSIOLOGY.

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## BLOOD.

**Quantity of Blood in the Body.**—Haldane and Lorrain Smith [1] have determined the total mass of the blood in man by causing the person to inhale a known volume of carbonic oxid mixed with oxygen and then determining in a sample of blood the percentage amount to which the hemoglobin has become saturated with CO. From this the amount of CO capable of being taken up by the whole of the blood can be calculated. All that remains is to estimate the volume of CO (or, what is precisely the same thing, the volume of O) which 100 gm. of blood will take up (that is, the percentage oxygen capacity). From these data we get at once the quantity of blood in the body, which, according to Haldane and Smith, has hitherto been greatly overestimated. They find that it is, on an average, only about 4.9 % ($\frac{1}{20.5}$) of the body-weight, varying in 14 persons between 3.34 % ($\frac{1}{30}$) and 6.27 % ($\frac{1}{16}$) of the body-weight. In chlorosis and pernicious anemia Smith [2] states that the quantity of the blood is markedly increased ; in one case of pernicious anemia to $\frac{1}{7.2}$ of the body-weight. This is due to the increase in the plasma. The **total oxygen-capacity** (*i. e.*, the total quantity of O which the blood will take up) is, according to the same authors, in a normal individual about 0.85 liters for 100 kilos of body-weight. It is but little altered in chlorosis, but is markedly diminished in pernicious anemia. In both diseases the percentage oxygen-capacity is greatly lessened, owing to the decrease in the number of red corpuscles and in the amount of hemoglobin. [Gréhant and Quincke have previously employed the inhalation of CO to determine the quantity of blood in the dog, and, comparing it with Welcker's method, have found that it yielded accurate results. It is clear that the whole method rests on the assumption that CO is not to any appreciable extent destroyed in the body during the period of the experiment. Various observers have been led to the contrary result, among others Wacholtz,[3] whose experiments, however, we adversely commented upon in a previous report.[4] Haldane,[5] after repeating Wacholtz's work, now rejects it altogether, and finding no evidence that CO is oxidized in the body, reiterates the accuracy of his method, which appears to be based on sound principles.]

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[1] Jour. Physiol., vol. XXV, p. 331 ; Proc. Physiol. Soc., p. v.

[2] Proc. Physiol. Soc., p. vi.

[3] Pflüger's Arch., Bd. LXXIV, S. 174 ; Ibid., Bd. LXXV. S. 341.

[4] YEAR-BOOK for 1900, Medicine, p. 506.

[5] Jour. Physiol., vol. XXV, p. 225.

**Osmotic Relations of Blood.**—Oker-Blom,[1] in a series of elaborate researches on the electric conductivity and general osmotic relations of the corpuscles and plasma, confirms the conclusion of Roth,[2] Stewart,[3] and Bugarsky [4] that the red corpuscles are nonconductors in comparison with the plasma, and the results of Stewart on the effect of dilution of the blood on its conductivity. A. Rollett [5] also confirms the conclusions of the latter [6] as to the effects produced when blood is laked by various reagents, and especially his statement that the hemoglobin and electrolytes may be caused to leave the corpuscles independently [a fact of considerable interest in connection with the question of the relation of the hemoglobin to the stroma of the corpuscles.] Rollett sees in his work a further proof of the correctness of his view that the corpuscle is not a mere vesicle, but contains a framework of semisolid stroma. [And, indeed, we do not believe that the contrary theory can be any longer maintained in face of the chemicophysical results of the past two or three years.]

**Coagulation of Blood.**—W. H. Thompson [7] has shown that the protamins (substances hitherto obtained only from the ripe milt of certain fishes and believed by Kossel to be the simplest proteids) exert, when injected intravenously, a retarding influence on coagulation, diminish the number of leukocytes, and lower the blood pressure, just as albumoses do. They also affect the respiration, causing first exaggeration, then a gradual diminution, and then cessation. These effects are partly due to a direct depressing action on the respiratory muscles.

## CIRCULATION.

**Action of Na, Ca, and K Ions on the Heart.**—An important series of papers has been published by J. Loeb on the effect of various ions on rhythmic contraction in general and particularly on the rhythmic contraction of the heart. He starts with the observation [8] that a striated muscle in a $\frac{1}{8}$ normal NaCl solution (or in the equimolecular solution of any other Na salt) may go on contracting rhythmically for from 24 to 48 hours, while Ca and K ions only inhibit these contractions. Nevertheless the muscle remains longer alive when a small amount of $CaCl_2$ and KCl is added to the NaCl solution. Loeb explains the seeming paradox by the hypothesis that the Na ions are the real stimulus for the rhythmic contractions, but yet exert on the muscular tissue a poisonous influence, which is neutralized by the Ca and K ions. He finds support for the idea that the Na ions are actually poisonous to the living substance in the fact [1] that *Fundulus heteroclitus* —a small marine fish, with so marvelous a range of adaptation that it will live in sea-water to which NaCl has been added to the amount of 5 %, and, on the other hand, in fresh and even in distilled water,[2]—will not live in pure NaCl solutions of about the same osmotic pressure as sea-water, but will survive in NaCl solutions even twice as strong if a little $CaCl_2$ or KCl be added. The K ions are, therefore, only indirectly necessary in order to neutralize the poisonous action of the NaCl. In another paper [3] he extends his conclusions as to the poisonous action of Na ions and the protective action of Ca and K ions to many other marine animals, and defends it against the obvious objection that, according to current physiologic doctrine, the Ca and K ions are indispensable for the contraction of the cardiac muscle. As a matter of fact, according to J. Lingle,[4] one of Loeb's pupils, a pure NaCl solution has an injurious effect upon the ventricular muscle of the turtle, while Ca and K ions improve the rhythm by neutralizing this injurious action. The reason for all these peculiar effects of the Na, Ca, and K ions Loeb finds in the formation by them of combinations with the proteids of the living substance, which he imagines to remain contractile only so long as the combination contains all three classes of ions in proportions varying within certain definite limits. In a pure NaCl solution the Na ions get the upper hand, and the balance is upset. He even goes so far as to say that the normal proportions of Na, Ca, and K ions vary in different kinds of tissues, as in muscle and nerve, so that in Gonionemus (one of the Hydromedusæ), *e. g.*, myogenic contractions are prevented by a smaller amount of K and Ca ions in the surrounding NaCl solution than neurogenic contractions originating in parts containing ganglia.

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[1] Pflüger's Arch., Bd. LXXIX, S. 111; Ibid., S. 510; Ibid., Bd. LXXXI, S. 167.

[2] Centralbl. f. Physiol., Bd. XI, July 10, 1897.

[3] Ibid., Aug. 7, 1897; Jour. Bost. Soc. Med. Sci., June 3, 1897; Jour. Physiol., vol. XXIV, p. 211.

[4] Centralbl. f. Physiol., July 24, 1897.

[5] Pflüger's Arch., Bd. LXXXII, p. 199.

[6] YEAR-BOOK for 1899, p. 950; Ibid. for 1900, p. 501.

[7] Zeit. f. physiol. Chem., Bd. XXIX, S. 1.

[8] Fick's Festschrift, Braunschweig, 1899.

**Influence of Chloroform on the Heart.**—J. A. MacWilliam,[5] whose previous accurate researches on this subject have so much advanced our knowledge, returns to the question in a paper on the effect of the drug on the rate of the pulse. He states that in the cat, as in man, there is first a stage of acceleration and then a stage during which the heart beats more slowly than normal. The acceleration essentially depends on a diminution in the activity of the vagus center; and the subsequent slowing, partly on increased activity of that center and partly on a direct action of the drug on the heart itself, not connected with the changes in the blood pressure. An important practical point is that at a certain stage in chloroform anesthesia, before it has become very deep, comparatively trifling causes may bring about great and sudden changes in the pulse-rate, owing to the abnormal mobility of the vagus center. Sudden arrest of the respiratory movements causes effects that vary at different stages of the anesthesia, but always terminate in a phase of slowing and irregularity.

**Tetanus of the Heart.**—[Few questions in physiology have been more generally discussed than the possibility of causing a true tetanus of the heart. That it is difficult to do so under ordinary conditions is admitted by all, and most systematic writers have explained the difficulty as due to the long refractory period of the cardiac muscle.] E. v. Cyon [1] believes that he has discovered evidence that the phenomenon is not related to any peculiar property of the muscle of the heart, but to the existence in its walls of automatic nerve-centers, motor and inhibitory, which, so long as they are acting in normal coordination, hinder the achievement of complete tetanus. [We must say that Cyon's explanation is rather a nebulous one, but, such as it is, he finds support for it in the fact that a frog's heart which has been brought to a standstill by heating it to 40° C., and in which, according to him, the inhibitory apparatus has been put out of gear, can be tetanized by stimulation of the vagus. But while it may be considered certain that the refractory period of the heart is diminished and its capacity for entering into tetanus increased by raising the temperature, we can not accept Cyon's view that stimulation of the inhibitory fibers has anything to do with the outburst of very rapid contractions caused by excitation of the frog's vagus during heat-standstill of the heart. On the contrary, it was shown long ago by the author of this abstract [2] that the phenomenon was due to stimulation of the sympathetic augmentor fibers in the nerve.] The experiments of O. Frank [3] and Rouget,[4] who obtained tetanus of the heart by simultaneous excitation of the vagus and sinus venosus, and those of A. Walther,[5] who obtained it by exciting a heart poisoned with muscarin with strong interrupted currents, are much more conclusive than those of Cyon, and indicate that there is some relation between the inhibitory mechanism and the production of tetanus.

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[1] Pflüger's Arch., Bd. LXXX, p. 229.

[2] Ibid., Bd. LV.

[3] Am. Jour. Physiol., vol. III, p. 327.

[4] Am. Jour. Physiol., vol. IV, p. 265.

[5] Jour. Physiol., vol. XXV, p. 233.

**The Intersystole of the Heart.**—[It has often been debated whether any appreciable interval exists between the end of the auricular and the beginning of the ventricular systole of the warm-blooded heart. According to Chauveau,[6] not only is this period (the intersystole) well marked and sharply delimited (in the horse), but it is occupied by a definite series of events. In particular, the contractions of the papillary muscles produce changes during this period in the interior of the ventricles. He refers [in gently ironical terms] to the recent work of Potain,[7] who could find no intersystole at all, and finally dismisses it [as we can] with the remark that Potain's results must have been due to imperfect technic.

**Electric Changes in the Heart.**—W. Einthoven and K. de Lint [8] have published some beautiful records [the best we have seen] of the electric changes that accompany the beat of the human heart. They show that exercise has a marked effect on the electrocardiogram, which is, moreover, altered in disease (aortic insufficiency). P. Rivière,[9] after a re-investigation of the question whether the electric variation of the heart during a single contraction is multiple,—*i. e.*, made up of a series of oscillations, as in experimental tetanus,—comes to the conclusion [which is the orthodox and, we believe, the true one] that it is a single variation. [When apparently multiple oscillations have been seen, they are either abnormal or due to imperfections in the method of recording.]

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[1] Jour. de Phys. et de Path. gén., tome II, p. 395; Ibid., p. 644.
[2] Jour. Physiol., XIII, p. 59.
[3] Zeit. f. Biol., Bd. XXXVIII, p. 300.
[4] Arch. de Physiol. norm. et path., 1894.
[5] Pflüger's Arch., Bd. LXXVIII, p. 597.
[6] Jour. de Phys. et de Path. gén., tome II, p. 125.
[7] Jour. de Phys. et de Path. gén., tome II, p. 101.
[8] Pflüger's Arch., Bd. LXXX, S. 139.
[9] Jour. de Phys. et de Path. gén., tome II, p. 275.

**Output of the Heart.**—[All physiologists are now agreed that the output of the ventricle was vastly overestimated by the older observers, such as Vierordt and Volkmann.] Cowl,[1] who has studied the changes of volume of the heart during the cardiac revolution by means of Röntgen radiographs, supports the conclusion of Zuntz and other recent investigators that the output is probably, on the average, in the neighborhood of 50 cc. or 60 cc. Notwithstanding the movements of the heart, he gets sharp photographs, since, as he estimates, the actual variation in the breadth of the organ is only 8 mm. on a total transverse diameter of 90 mm.

**Pulsus Bigeminus and Trigeminus.**—[The rapid development of the physiology of the mammalian heart is at last beginning to yield fruit in the solution of clinical problems of long standing.] H. E. Hering,[2] on the basis of careful experiments on animals, essays the interpretation of the irregularities in the pulse due to increase of resistance to the emptying of the left or right ventricle, which express themselves as a pulsus bigeminus or trigeminus. They are caused, according to him, by an alteration in the normal sequence of the auricular and ventricular contractions, the interval between the systole of the auricle and that of the ventricle becoming smaller, or equal to nothing, or even negative. In the last case a complete reversal of the normal sequence may take place. This alteration in the sequence has its origin in the appearance of premature ventricular contractions brought about by the excitation of the ventricle, not in the normal way from the auricle, but, before the arrival of the auricular excitation, by an abnormal mechanical stimulus acting directly on the ventricle and due to the unusual resistance to discharge. The pulsus bigeminus can also be induced by artificial stimulation of the auricle or ventricle. But while the time-value of a bigeminal pulse elicited by excitation of the ventricle is equal to that of two regular heart-beats, this is not the case when the bigeminus is due to stimulation of the venæ cavæ, and is the case only under certain conditions with a bigeminus caused by stimulation of the auricle. One can therefore to a certain extent determine from the time-value of a bigeminus what part of the heart is affected by the pathologic condition. [Several of Hering's conclusions, and especially the relation of the time-value of the abnormality in the pulse with the portion of the heart responsible for it, were clearly formulated by Cushny[3] for the intermittent pulse a year before the appearance of Hering's paper. Wenckebach [1] also indicated this relation, although not so precisely, in a still earlier publication, basing his conclusion on the experimental results of Cushny and Matthews [2] on the dog's heart.

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[1] Ver. d. Berl. physiol. Ges.; Arch. f. (Anat. u.) Physiol., p. 564, 1900.

[2] Pflüger's Arch., Bd. LXXXII, S. 1.

[3] Jour. of Exp. Med., vol. IV, p. 327.

**Velocity of Venous Pulse.**—W. S. Morrow [3] has measured the velocity of the venous pulse which propagates itself centrifugally through the veins of the trunk and extremities from the right auricle and the venæ cavæ. It varies between 1 and 3 meters a second, and is at most only half as great as the velocity of the arterial pulse.

**Depressor Nerve.**—W. T. Porter and H. G. Beyer [4] find no sufficient evidence that the depressor nerve (in the rabbit) [as many authors assume] is specially connected with the cells which control the vasomotor fibers of the splanchnic nerves; for when the normal blood pressure is restored after section of the splanchnics,—*e. g.*, by the injection of warm normal saline solution into the jugular vein,—stimulation of the central end of the depressor causes usually as great a fall of pressure as when it is stimulated while the splanchnics are intact. They accordingly suppose that the depressor nerves are probably related in the same way to all the cells of the bulbar vasomotor center, so that their excitation brings about general dilation of the arterioles throughout the body. [There need, we think, be no great hesitation in accepting this conclusion, both on account of the experimental evidence by which it is supported and on account of its intrinsic probability. For the depressor nerve, from its special relation to the heart, may be supposed to aid in keeping the general blood pressure within physiologic limits rather than to take part in any more direct manner in regulating the distribution of the blood to particular organs. But few, we imagine, will be prepared, without special proof, to accept the further suggestion of these observers that all afferent vasomotor fibers may be similarly connected with all the nerve-cells of the general vasomotor center, so that the bulbar center would have no share at all in the distribution of the blood, but would only provide for a general rise or fall of pressure.]

## RESPIRATION.

**Section of the Vagi.**—[Since Béclard's time it has been stated that if a considerable interval be allowed to elapse after section of one vagus before the other is divided the animal may survive. The common explanation is that regeneration of the first vagus has taken place before the second is cut.] R. Nicolaides [5] records some experiments in which, at least 45 days after the resection of 5 cm. of one vagus in the neck, a piece of equal length was cut out of the other. In all, 3 dogs were operated upon, of which 1 died on the third day, while 2 were alive at the time the paper was written, one 92 days, and the other 22 days, after the operation. [We do not see anything surprising in these results, as this observer seems to do. Such cases of long survival have been observed by the author of this abstract even when both vagi were divided in the neck at the same time. Among 30 dogs operated on there was one which would apparently have survived indefinitely, and another in which death occurred nearly 5 weeks after the operation. On the other hand, in a dog whose second vagus was divided 10 months after the first, death ensued 3 days after the second operation. There are great individual differences in the resistance of dogs to double vagotomy, and much care is necessary in drawing conclusions as to the cause of the differences in the period of survival. It is certainly impossible to deduce any general law from the results of 3 experiments.]

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[1] Zeit. f. klin. Med., Bd. XXXVI, Hefte 3 u. 4, 1898; Ibid., Bd. XXXVII, Hefte 5 u. 6, 1899; Nederl. Tydschr. v. Geneesk., 1898, T. II; Ibid., 1899, T. I, No. 16, p. 655; T. II, No. 24, p. 1132.

[2] Jour. Physiol., vol. XXI, p. 213.

[3] Pflüger's Arch., Bd. LXXIX, S. 442.

[4] Am. Jour. Physiol., vol. IV, p. 283.

[5] Centralbl. f. Physiol., Bd. XIV, S. 197.

**Respiratory Exchange in the Embryo.**—[The observations of Zuntz and Cohnstein [1] on the relative amount of the blood-gases in the umbilical arteries and vein have been generally accepted as an exact proof of Pflüger's dictum that the metabolism of the embryo is much smaller in proportion to its body-weight than that of the adult. Bohr and Hasselbalch,[2] however, using a more accurate method, have convinced themselves that this idea is erroneous, and that the production of carbon dioxid both in the embryo fowl and the embryo mammal (guinea-pig) is of the same order of magnitude as in the adult. [In the light of these experiments, which appear to have been carried out with great care, and to have yielded sufficiently uniform results, the current estimate of the intensity of embryonic metabolism certainly appears to need revision. The explanation of its magnitude, at first thought so surprisingly great, is probably to be sought, as Gusserow [3] first pointed out, in the rapid growth of the embryo, which can hardly be supposed to go on without a considerable metabolism.]

Levy and Falk,[4] in a very elaborate paper, including many valuable tables, communicate the results of their researches on the **gaseous exchange in man at different ages.** They place upon a firm foundation the popular opinion that the gaseous metabolism of children per unit of weight is greater than in the adult and greater in the inverse proportion of the age and body-weight. For the first year of life, however, this does not hold. In old age the metabolism per kilo sinks. In the female the gaseous exchange per unit of body-weight [contrary to the ordinary statement] is the same as in the male.

The contradictory results of previous workers as to the relative importance of the **pulmonary and cutaneous respiration in the frog** have been explained [very plausibly, we think] by C. Bohr,[5] who finds that the effect of exclusion of the lungs depends on the previous intensity of the metabolism. If this is high, the gaseous exchange sinks markedly; if it is low, there is scarcely any alteration in it. The lungs are capable of sustaining a much greater exchange than the skin. In addition to this quantitative, there is a qualitative difference, the carbon dioxid passing more easily through the skin than the oxygen, while the two gases pass with equal ease through the pulmonary membrane. The respiratory quotient is accordingly increased when the lungs are eliminated.

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[1] Pflüger's Arch., Bd. XXXIV, S. 173.

[2] Skand. Arch., Bd. X, S. 149; Ibid., S. 353; Ibid., S. 413.

[3] Arch. f. Gynekol., Bd. III, S. 241.

[4] Arch. f. Physiol., Supp. Bd., S. 314, 1900.

[5] Skand. Arch., Bd. X, S. 74.

## DIGESTION AND ABSORPTION.

**The Soft Palate.**—A. Couvelaire and O. Crouzon,[1] without perhaps advancing anything materially new, have added precision to our knowledge of the rôle of the soft palate in deglutition, respiration, and phonation, by a careful study of a case of naso-orbital deficiency due to an old surgical operation. Among other facts, they have actually observed that complete occlusion of the nasopharynx is caused in deglutition, sucking, whistling, and during muscular effort by the projection of the posterior and lateroposterior wall of the pharynx, which comes into contact with the free border of the soft palate. In coughing the occlusion is incomplete. It is variable in phonation, the palate taking up a particular position for each vowel.

**Sphincter of Anus.**—L. v. Frankl-Hochwart and A. Fröhlich[2] have made an exhaustive investigation of the tone and innervation of the anal sphincters in the dog. They conclude that at the lower end of the rectum there exists a tonic sphincter mechanism independent of the will, but governed by the nervous system. This is shown by the fact that in life a certain resistance is experienced here by a stream of water. This resistance almostly completely disappears after the administration of certain poisons or the resection of the peripheral nerves supplying the part. While the internal sphincter is by itself capable of maintaining a tonus of considerable strength, the external sphincter contributes an important share (30% to 60%) to the closure of the rectum. As to the nervous mechanism, the nervi erigentes carry efferent constrictor fibers and the hypogastrics, as a rule, efferent dilator fibers, to the sphincters. A reflex center exists in the cord, but even after this is destroyed tonic constriction can be brought about reflexly through certain peripheral nerves, the precise identification of which is left in doubt.

**Rectum.**—[A difference of opinion exists as to whether the rectum is empty in the intervals between successive acts of defecation, these being excited by the passage of feces into it from the sigmoid flexure, or is gradually filled with feces, the distention produced by which, when it reaches a certain limit, gives rise to the reflex stimulation that normally initiates the act.] J. J. Charles[3] brings together the arguments in favor of the latter view, which he himself adopts. [While nobody who has made a large number of digital examinations of the rectum can doubt that the lower end of it is sometimes filled with feces, there is no evidence that this is usually the case. And nothing is more likely than that in persons who habitually neglect the call to stool the rectum should become less sensitive to the entrance of the first portions of feces from the sigmoid flexure than in those who promptly obey it.]

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[1] Jour. de Phys. et de Path. gén., tome II, p. 280.

[2] Pflüger's Arch., Bd. LXXXI, S. 420.

[3] Brit. Med. Jour., Sept. 30, 1899; N. Y. Med. Jour., Nov., 1899.

**Formation of HCl in the Stomach.**—[Koeppe's theory that the HCl of the gastric juice is formed from the chlorids of the gastric contents and not from the chlorids of the blood has been vigorously attacked both on theoretic and experimental grounds, and seems hardly to be holding its own.] The last paper on the subject by J. A. Wesener[1] brings fresh evidence against it. He washed out the stomach of a fasting individual so as to remove the chlorids and HCl, and then caused a secretion of gastric juice by mechanically stimulating the mucous membrane by means of Turck's "Gyromele."[2] The gastric contents, which before were neutral, now contained a considerable quantity of free HCl.

**Pancreatic Juice.**—B. K. Rachford, between whom and Chittenden and Albro a controversy has been carried on with regard to the influence of bile on the proteolytic action of the pancreatic juice, returns to the charge in a series of new experiments,[3] in which he believes he has shown, in opposition to the statement of these observers, that when fresh rabbit's bile is added to fresh pancreatic juice acting on neutral fibrin, the proteolytic power of the juice is markedly stimulated. [This conclusion seems to be correct, and is supported by the observation of Zuntz and Ussow[4] that ox bile, and in a slighter degree the pure bile-salts, hasten the tryptic action of ox pancreas on proteid, as well as its amylolytic action on starch.]

**Reaction of Intestinal Contents.**—[B. Moore and D. P. Rockwood[5] have previously shown that much of the confusion which reigns in regard to this question is due to the fact that various indicators have been indiscriminately used without sufficient attention being paid to the manner in which each is affected by such substances as are present in the alimentary canal.] Moore and T. J. Bergin[6] make a further contribution to the subject by showing that the acid reaction of the intestinal contents to phenolphthalein is probably due to an excess of dissolved $CO_2$, while the alkaline reaction to methyl-orange, lacmoid (litmoid), and litmus shows the absence of HCl and of all stronger organic acids (such as acetic, lactic, or butyric) which would be formed in the bacterial decomposition of carbohydrates or fats. [These experiments seem to have been done mainly on animals engaged more or less actively in digestion. In dogs after digestion is over (20 to 24 hours after a meal) the reaction has been found by Pluemer and Stewart[7] to be acid to methyl-orange, litmus, and phenol-phthalein throughout the whole intestine. The meaning of this may be that toward the end of intestinal digestion, the discharge of bile, pancreatic juice, and succus entericus having almost or entirely ceased, the acid-forming bacteria again get the upper hand.]

**Xanthin Bases in Feces.**—The solution of the question whether the xanthin bases known to exist in normal human feces come directly from the constituents of the food or are produced in the metabolism of the tissues has been advanced a stage by the observations of K. Petrén [1] and W. H. Parker.[2] The former found that xanthin bases were present in the feces of a patient fed exclusively on milk. Now, xanthin bases can not be obtained from milk,[3] and therefore in the case investigated they must have come from the body. Parker likewise obtained xanthin bases in the feces when the diet contained no nucleins; but, on the other hand, he saw an increased excretion when a diet rich in alloxuric bodies (such as meat extract or thymus) was given. It is, therefore, probable that the xanthin bases of the feces, under ordinary circumstances, come both from the food and from the tissues.

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[1] Pflüger's Arch., Bd. LXXVII, S. 483.

[2] YEAR-BOOK for 1897, p. 155.

[3] Jour. Physiol., vol. XXIV, p. 165.

[4] Ver. d. Berl. physiol. Ges.; Arch. f. (Anat. u.) Physiol., p. 380, 1900.

[5] Jour. Physiol., vol. XXI, p. 58.

[6] Am. Jour. Physiol., vol. III, p. 316.

[7] Manual of Physiology, by G. N. Stewart, 4th ed., p. 353, 1900.

**Absorption of Fat.**—[As was stated in a previous report [4] many voices have of late years been raised against the common assumption that the greater part of the fat is absorbed as neutral fat in the form of an emulsion. Several of the more recent observers, indeed, including E. Pflüger,[5] go so far as to say that none of it is so absorbed, but that the whole is first split up into fatty acids and the corresponding alcohol. I. Munk [6] protests against this extreme view. He believes that only a portion of the fat is absorbed in the soluble form, and brings forward as a decisive argument in favor of the emulsion theory the undoubted fact that after extirpation of the pancreas a considerable quantity of fat still continues to be taken up from the intestine. [But it scarcely requires to be pointed out that this fact is decisive only on the assumption that no fat-splitting can take place in the intestine in the absence of the pancreatic juice—an assumption which is so far from being proved that the best observations (those of Minkowski, *e. g.*) point emphatically to the conclusion that fats can not only be split up after the pancreas has been removed, but that the amount of such fat-splitting is astonishingly great.] Nor can we attribute any greater value for the decision of this question to the result of R. H. Schmid, quoted by Munk, that liquid fats quickly find their way into vegetable cells, as is seen when the fat is colored with alcanna red, nor to the observation of L. Hofbauer [7] that in dogs fed with fat colored with alcanna and "lackroth A" colored fat is found in the lacteals and epithelial cells of the intestine, and that in a case of chyluria when fat tinged with "sudan red III" was added to the diet of the patient the previously milk-white urine became rose-colored; for E. Pflüger,[8] H. Friedenthal [9] and V. Henriques, and C. Hansen [10] have shown that these pigments are not suitable for such experiments, since they are all soluble in soaps, in free fatty acids, and in other substances, as bile and glycerin, which are present in the intestinal contents. Henriques and Hansen,[11] using mixtures of paraffin and fat, which can be emulsified in a watery solution of sodium carbonate, have demonstrated that the paraffin is completely excreted with the feces, while the greater part of the fat is absorbed. They conclude that the fat in their experiments can have been absorbed only in the soluble form (as soaps). [As we remarked last year [1] with reference to Cohnstein's observations on the absorption of lanolin, this conclusion seems the logical one, unless we suppose that the intestinal epithelium *selected* the fat out of the mixture and rejected or re-excreted the paraffin as useless to the economy —an assumption for which there is no evidence.]

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[1] Skand. Arch., Bd. VIII, S. 412.

[2] Am. Jour. Physiol., vol. IV, p. 83.

[3] Petrén, Skand. Arch., Bd. IX, S. 412.

[4] YEAR-BOOK for 1900, p. 509.

[5] Pflüger's Arch , Bd. LXXXII, S. 303.

[6] Centralbl. f. Physiol., Bd. XIV, S. 121; Ibid., S. 153.

[7] Pflüger's Arch., Bd. LXXXI, S. 263.

[8] Pflüger's Arch., Bd. LXXXI, S. 375.

[9] Centralbl. f. Physiol., Bd. XIV, S. 258.

[10] Centralbl. f. Physiol., Bd. XIV, S. 313.

[11] Loc. cit.

**Path of Absorption of Fat.**—[It is usually stated in the text-books that practically the whole of the fat is absorbed by way of the lacteals. It has, however, been long suspected that a not inconsiderable portion may enter the rootlets of the portal vein, for it is never possible to recover from the lymph of the thoracic duct the whole of the fat taken up from the intestine. It has been mainly the supposed difficulty of understanding how undissolved fat can find its way through the capillary walls which has enabled the current view to hold its ground in spite of this deficiency. But if it be granted that the fat is altogether or in great part absorbed in a soluble form, this difficulty vanishes; and it is curious to observe that the change of theoretic standpoint which we have discussed in the preceding paragraph has been followed by a change in the tenor of the experimental results.] H. J. Hamburger,[2] *e. g.*, after tying the lacteals of a loop of intestine, saw a considerable absorption of emulsified olive oil. According to the same observer,[3] contrary to the common opinion, considerable quantities of fats can be absorbed from the large intestine (of the dog) when injected in the form of an emulsion which maintains itself for a considerable time in the intestine. A solution of soap is the best medium for the fat; and the soap is also itself absorbed, being, in part at least, changed into fat in the mucosa. [The physiologic reason for the synthesis of soaps to fats in the intestinal wall is an interesting subject of speculation. I. Munk's experiments led him to the idea that soaps might be poisonous if present in the blood in more than a certain amount, since he always found that the quantity of soaps in the blood-plasma varied within narrow limits. On testing the question further,[4] he found that, as a matter of fact, soaps, when injected intravenously in the rabbit, did exert a poisonous action.] P. Botazzi,[5] who has recently repeated these experiments, confirms Munk as to the facts, but differs as to the explanation. He suggests that the poisonous action is not due to the soaps as such, but to the alkali which is split off from them in the body. Munk,[6] however, in a second paper shows that Botazzi's theory is erroneous, and that the toxicity is a property of the soaps.

On the **general problem of absorption** the most important papers of the year are those of Waymouth Reid [1] and H. Friedenthal.[2] Reid here sums up the results of much of his previous work, some of which we have already noticed in a former report.[3] He sees in the intestinal epithelium the seat of the forces through which what we speak of in common parlance as the selective activity of the wall of the intestine is manifested. These cells are also responsible for the orientating action of the intestinal mucosa on salts in solution (which was first pointed out by O. Cohnheim);[4] that is to say, for the kind of action in virtue of which under normal circumstances salts tend rather to pass into the blood from the lumen of the intestine than from the blood into the lumen. [Both Reid and Friedenthal conclude that a certain portion of the phenomena of absorption can be shown to depend upon known physical forces. But the Scotch observer recognizes more clearly than his German colleague how large a residue there is which still remains unexplained and even unexplored, and with the proverbial and, in matters scientific, invaluable caution of his nation, stops short where his knowledge stops and refuses to generalize beyond the facts. This attitude of reserve is in no part of physiology more necessary than in dealing with the subject of absorption, which has too long been the sport of theory.]

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[1] YEAR-BOOK for 1900, p. 509.

[2] Arch. f. (Anat. u.) Physiol., S. 554, 1900.

[3] Arch. f. (Anat. u.) Physiol., S. 433, 1900.

[4] Centralbl. f. d. Med. Wiss., No. 28, 1889; Arch. f. (Anat. u.) Physiol., Supp. Bd., S. 116, 1890.

[5] Arch. ital. de Biol., tome XXXII, p. 176.

[6] Centralbl. f. Physiol., Bd. XIII, S. 657.

## METABOLISM AND DIETETICS.

**Nitrogen Equilibrium.**—[The fact that the body can remain in nitrogen equilibrium with very different amounts of proteid in the food is one of the perennial puzzles of physiology.] E. Pflüger [5] attempts to find an explanation of the apparently useless destruction of proteid in the circumstance that an increase of proteid in the food causes an increase in the working power of the organism, accompanied, of course, by an increase in the metabolism, and also an increase in the body-weight, owing to the growth of the cell-substance. The metabolism and working power of the body increase in exact proportion to the increase in the body-weight caused by proteid, and the greatest metabolism and highest efficiency of the bodily machine can therefore be obtained only with the most plentiful supply of proteid in the food. Every diminution in the daily allowance of proteid is accompanied by a diminution in the metabolism and working power, even when fat and carbohydrates are given in place of the proteid withdrawn. [While the utterances of so distinguished a physiologist must always command attention we may, without offense, characterize this paper as the *ne plus ultra* of the proteid-worshiping school. Through it all, we can not help thinking, there runs a certain note of exaggeration somewhat foreign to the style of the author of "Electrotonus," or at least an over-emphasis that easily slips into fallacy. The importance of, indeed, the absolute necessity for, a certain amount of proteid is one of the commonplaces of the science. But every-day experience seems to show that the highest mental and physical efficiency is compatible with a relatively low proportion of proteid in the diet.] This is borne out by such careful experimental results as those of Sivén,[1] who, in an attempt to fix the minimum quantity of proteid required, found that, for a short time at any rate, an adult, without increasing the total heat-value of the food beyond the normal, can remain in nitrogen equilibrium and good working condition with an income of nitrogen of only 4.52 gm. (= 0.08 gm. N per K of body-weight), an amount equivalent to 28.3 gm. proteid. Only 2 gm. of this N need be the N of pure albumin. The greatest amount of N in any of his experiments was 12.69 gm. a day. He concludes [rightly, we think] that the proteid in the daily food of a strong man doing hard work can be reduced much below 118 gm., the amount given by Voit as the standard for the average workman. There is no real inconsistency between this conclusion and such results as those of K. Ekholm,[2] who, in a paper containing an excellent summary of previous work in this field, states that a considerable number of soldiers investigated by himself, and of students investigated by Tigerstedt, when allowed to eat as much as they liked of a given diet and to do no muscular work, consumed quantities of proteid whose heat-equivalent amounted to very much the same fraction of the total heat-value of the food as the heat-value of the proteid in Voit's standard dietary does.

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[1] Phil. Trans., vol. CXCII, B., 1900; Brit. Med. Jour., June 30, 1900.

[2] Arch. f. (Anat. u.) Physiol., S. 217, 1900.

[3] YEAR-BOOK for 1899, p. 958.

[4] YEAR-BOOK for 1899, p. 958; Ibid. for 1900 (Medicine), p. 509.

[5] Pflüger's Arch., Bd. LXXVII, S. 425.

**Production of Fat and Glycogen from Proteid.**—E. Pflüger[3] reasserts his view, in opposition to the Munich school, that fat is not formed from proteid, and endeavors to show that the carbon kept back in the body on a plentiful diet of proteid may be represented by leucin and tyrosin and not by fat at all. His pupil, B. Schöndorff[4] (with H. Offergeld), further emphasizes the distinction between proteid and other food-substances by what he believes to be an absolute demonstration that no glycogen is formed from proteid when the latter is completely free from carbohydrate groups. For example, none is formed from pure casein. According to him, all statements to the contrary are based on experiments in which, in addition to proteids, carbohydrates were, or may have been, present in the food.

**Glycogenesis.**—C. Bouchard and A. Desgrez[5] attempt [not with complete success] to establish a difference of origin between the hepatic and the muscular glycogen, the former, according to them, coming from the carbohydrates and proteids; the latter, from the incompletely oxidized fats and in lesser degree from the sugar of the blood. [This statement will scarcely be accepted in its entirety in the absence of definite proof of the possibility of the conversion of fat into glycogen, which has not yet been given. There is, however, no reason to doubt that sugar may be changed into glycogen in the muscles.] L. Popielski,[6] on the basis of experiments in which he fed with known quantities of sugar dogs whose inferior vena cava and portal vein had been united by an Eck's fistula, and determined the amount which passed into the urine, estimates the quantity of sugar kept back by the liver at from 12% to 20% of the whole. The rest passes on into the general circulation and is partly burnt in the tissues, partly kept back in the muscles and other organs. [The apparently endless controversy as to whether sugar is normally formed in such amount in the liver that the blood of the hepatic vein in the fasting animal is appreciably richer in that substance than the blood of the portal vein still drags its weary length along, Seegen,[1] *e.g.*, on the one hand, maintaining that there is no doubt that this is the case, while Bing[2] and others state that no difference can be with certainty detected. Incidentally, Seegen denies and Bing asserts the existence in the blood of jecorin, a reducing substance from which sugar can be obtained. As to this minor question, the weight of evidence is undoubtedly in favor of the presence of jecorin. On the main controversy, as to whether there is sufficient proof that the glycogen of the liver is normally reconverted into sugar *intra vitam*, we have expressed the opinion in several previous reports that such a conversion does take place, and we see no reason to alter it.]

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[1] Skand. Arch., Bd. x, S. 91.

[2] Skand. Arch., Bd. xi, S. 1.

[3] Pflüger's Arch., Bd. LXXVII, S. 521.

[4] Pflüger's Arch., Bd. LXXXII, S. 60.

[5] Jour. de Physiol. et de Path. gén., tome II, p. 237.

[6] Centralbl. f. Physiol., Bd. XIV, S. 193.

Seegen[3] has continued his researches on the preliminary stages of sugar formation in the liver from proteids, and, in addition to the nitrogenous body previously described,[4] has discovered another substance which can be changed into sugar by heating with dilute acid.

**Nutritive Value of Alcohol.**—[The alcohol question has come up in rather an acute form in the medical and physiologic journals during the past year.] W. O. Atwater[5] has made a series of estimations of the N and C excreted by a man living in the so-called respiration calorimeter previously described by Atwater and E. B. Rosa.[6] The person was fed with a diet of known composition, and received also known amounts of alcohol. The general result of the observations [which is that of most previous experimenters, including Zuntz] is that alcohol is burned in the body as completely as bread, meat, and other ordinary food substances, and in the same way. In the oxidation all the potential energy of the alcohol is converted into heat or muscular energy. The alcohol protects the material of the body from consumption as well as corresponding quantities of sugar, starch, or fat would do.

P. Bjerre[7] finds also that alcohol has a nutritive value, although he acknowledges that in general it has a tendency to economize fat and carbohydrates rather than proteids. R. Rosemann,[8] in a critique of the work of R. O. Neumann[9] and other observers who have granted to alcohol a proteid-sparing action, asserts that there is not the slightest justification for such a claim, and that, as Miura,[10] Schmid,[11] and Schöneseiffen [1] have stated, it has, in fact, been definitely disproved. T. R. Offer's [2] defense of the proteid-sparing power of alcohol suffers the same fate at the hands of Rosemann in another vigorous example of Teutonic criticism.[3] He admits, however [what we do not suppose any physiologist who approaches the question with an unbiased mind can possibly deny], that alcohol protects fats and carbohydrates from combustion. [Whether it protects proteids to the same extent must, we believe, be left to the decision of further experiment. That Atwater's conclusions on this point are not quite justified by his published results has been urged by Woodbury and Egbert.[4]] Schumburg,[5] on the basis of a long series of careful ergographic observations, asserts that alcohol appears not to be a food-stuff like the carbohydrates, which yield work by their combustion. It belongs rather to the same group as coffee, tea, cola nut, and maté, which exert a stimulating action only when a residue of true food-substances (fats, carbohydrates, or proteids) is present, but not when the body is completely exhausted.

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[1] Centralbl. f. Physiol., Bd. XIII, S. 593.

[2] Centralbl. f. Physiol., Bd. XIII, S. 689; Skand. Arch., Bd. IX, S. 336.

[3] Arch. f. (Anat. u.) Physiol., S. 292, 1900.

[4] YEAR-BOOK for 1900 (Medicine), p. 511.

[5] Bull. No. 69, U.S. Dept. of Agriculture.

[6] Bull. U. S. Dept. of Agriculture, No. 63.

[7] Skand. Arch., Bd. IX, S. 323.

[8] Pflüger's Arch., Bd. LXXVII, S. 405.

[9] Arch. f. Hygiene, Bd. XXXVI.

[10] Zeit. f. klin. Med., Bd. XX, 1892.

[11] Inaug. Dissert., Greifswald, 1898.

[A question which ought not to be confounded with that of the nutritive value of alcohol in the physiologic sense of the term is the question of its action on particular organs of the body or on the general health. Here also there still reigns considerable uncertainty.]

E. Kraepelin,[6] who has studied the general effects of small quantities of alcohol more carefully than any one else, finds that such doses favorably influence speech-utterance and the association of words and ideas in thought [a fact which is illustrated in the practice of many after-dinner speakers]. The reaction time is at first shortened, but afterward increased. The force of the cerebral discharges concerned in voluntary movement is intensified, while the resistance of the muscles themselves to fatigue is diminished. Caffein, on the other hand, really increases endurance. Upon the whole, the effect of even small doses of alcohol is not to increase the real mental power, although for a time the mental machinery may move more easily. The after-effects are injurious, and may last for many hours. The continued use of 60 gm. of alcohol per diem caused from day to day a gradual decrease in mental ability. Kraepelin sums up unfavorably even to the moderate use of alcohol. So does V. Horsley.[7] Bunge [8] even goes so far as to say that statistics suggest that tippling among women interferes with lactation and renders them unable to nurse their children. R. Rosemann,[9] however, shows, from experiments on the goat, that alcohol has no influence on the secretion of milk, and even when taken in large doses appears in it only in very small amount; and when taken in small doses, not at all.

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[1] Inaug. Dissert., Greifswald, 1899.

[2] Wien. klin. Woch., XII, No. 41.

[3] Pflüger's Arch., Bd. LXXIX, S. 361.

[4] Jour. Am. Med. Assoc., Mar. 31, 1900.

[5] Arch. f. (Anat. u.) Physiol., Supp. Bd., S. 289, 1900.

[6] Ver. d. 71ten Versam. deuts. Naturforscher u. Aerzte; Lancet, Nov. 18, 1899.

[7] Brit. Med. Jour., May 5, 1900.

[8] Deut. med. Zeit., Sept. 25, 1899; N. Y. Med. Jour., Oct. 21, 1899.

[9] Pflüger's Arch., Bd. LXXVIII, S. 466.

## INTERNAL SECRETION.

**Suprarenal, Thyroid, and Pituitary.**—E. v. Cyon [1] develops further his conception of the relations between the thyroid and pituitary body and the heart. He states [2] that mechanical pressure exerted in any way on the hypophysis causes marked slowing and strengthening of the heart-beat, with no fall or even with a slight rise of blood pressure. If the hypophysis is destroyed, pressure has no such effect. The opening of the cavity in which the hypophysis lies has (in dogs) the same effect as section of the vagi. We must, therefore, assume that in this cavity there is normally a pressure which brings about a tonic excitation of the intracranial vagus center. In the hypophysis, accordingly, we have a mechanism which protects the brain against an excessive supply of blood. Of more value at present than these somewhat fantastic speculations are the interesting facts observed by Cyon in a comparative study of the action of various extracts and drugs (iodothyrin, suprarenal extract, hypophysis extract, atropin) when allowed to circulate through the brain without reaching the heart and through the heart without reaching the brain. Intracranial injection of suprarenal extract caused a rise of blood pressure like intravenous injection, although a much smaller one. He concludes that suprarenal extract acts in the same way, with only a quantitative difference, on the peripheral and central ends of the vasomotor and cardiac nerves, and upholds the correctness of his view that the main action on the vasomotor mechanism is central, as against the assertion of Oliver and Schäfer that it is peripheral. Boruttau,[3] on the other hand, without denying that there is a slight action on the vasomotor center, supports the contention of Oliver and Schäfer [which, as already stated in more than one previous report, we believe to be correct], and Gottlieb[4] confirms the statement of these observers that the vessels of the isolated kidney are contracted by the extract. He also shows by experiments on the isolated mammalian heart that the extract directly excites the cardiac motor apparatus.

W. H. Bates,[5] in an article on suprarenal therapy, makes the statement that although the internal administration of the extract does not affect the normal heart or the normal blood pressure, an intermittent pulse is rendered regular and a weak pulse is strengthened by it.

Svěhla [6] has investigated the action of extracts of the thyroid, suprarenal, and thymus of human embryos and children, and has determined that in the embryo no active substances are present. The activity of the extracts increases with age. The active substance in the extract of the thymus which, when injected into the circulation, causes acceleration of the pulse and diminution of the blood pressure may still be present in that gland at the age of 40 years. In the ox, on the other hand, the three glands contain active substances even in embryonic life.

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[1] Pflüger's Arch., Bd. LXXVII, S. 215.

[2] Pflüger's Arch., Bd. LXXXI, S. 267.

[3] Pflüger's Arch., Bd. LXXVIII, S. 97.

[4] Arch. f. exp. Path. u. Pharm., Bd. XLIII, S. 286.

[5] Med. News, Mar. 24, 1900.

[6] Arch. f. exp. Path. u. Pharm., Bd. XLIII, S. 321.

B. Moore and C. O. Purinton [1] likewise find that the active principle and the chromogen are absent from the suprarenal in the human embryo and the child at birth. In connection with this, it is of interest that L. B. Mendel,[2] like previous observers, has been unable to detect iodin in the thyroids of new-born children, although it is present in the adult, not only in the thyroid proper, but in even larger amount in the accessory thyroids.

[A very large number of other papers on the suprarenal, thyroid, and pituitary have been published during the past year. The limits of our space will permit us to mention only a few.]

Auld [3] constantly observed distinct hypertrophy of the thymus and spleen in cats and dogs after extirpation of the suprarenals.

Boinet,[4] in very numerous experiments on the rat, saw congestion and hypertrophy of the spleen in the majority of the animals after removal of both suprarenals, in one-fifth of the cases hypertrophy of the thymus, and in a few cases hypertrophy and congestion of the thyroid. Some of the animals survived a long time (as much as 198 days). In young rats after extirpation of the spleen and both suprarenals the thymus was found hypertrophied. [Although these observations suggest that the spleen or thymus can take on the function of the suprarenals when the latter are removed, great caution must be exercised in interpreting such results; for the size of the thymus, spleen, and other organs of this class is not so constant that it is easy to determine whether hypertrophy exists or not. S. Vincent,[5] for instance, in a series of very careful observations on the effect of extirpation of the spleen on the lymphatic and hemolymph glands and the bone-marrow, which some investigators have supposed to hypertrophy after this operation, could never convince himself beyond a doubt that this was the case.]

Moore and Purinton,[6] from estimations of the minimum quantity of suprarenal extract (after removal of the proteids) which will produce an effect on the blood pressure, come to the conclusion that the active substance can not be Abel's epinephrin [7] or v. Fürth's suprarenin, because the necessary dose of these isolated bodies is markedly greater than that of the simple extract. The same observers [8] have noticed in some of their experiments on the removal of the suprarenals extensive antemortem coagulation in the auricles. They believe that this accounts for the fatal result in those cases where death rapidly occurs. The clotting is due to the stagnation of the blood in the relaxed auricles owing to the great diminution of blood pressure.

**Nervous Tissue.**—W. A. Osborne and S. Vincent,[9] following up the observation of Schäfer and Moore [1] and Schäfer and Vincent,[2] that intravenous injection of extracts of nervous substance (brain, spinal cord, sciatic nerve) causes a fall of blood pressure, find that the fall, although marked, is temporary. It may be obtained after the vagi have been excluded by section or atropin, and is due to direct dilation of the arterioles by the extract. They state further that although cholin is present in small amount in such extracts, it is not the active principle, as suggested by Mott and Halliburton,[3] who saw a fall of blood pressure produced by injection of cerebrospinal fluid from cases of general paralysis of the insane,[4] and were able to isolate cholin from the liquid.[5]

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[1] Am. Jour. Physiol., vol. IV, p. 57.

[2] Am. Jour. Physiol., vol. III, p. 285.

[3] Brit. Med. Jour., 1899, I, p. 1327.

[4] Gaz. hebdom. de méd. et de chir., Mar. 11, 1900.

[5] Proc. Physiol. Soc., Dec. 9, 1899; Jour. Physiol., vol. XXV, p. ii.

[6] Pflüger's Arch., Bd. LXXXI, S. 483.

[7] YEAR-BOOK for 1900 (Medicine), pp. 513, 569.

[8] Am. Jour. Physiol., vol. IV, p. 51.

[9] Jour. Physiol., vol. XXV, p. 283; Proc. Physiol. Soc., Ibid., p. ix; Brit. Med. Jour., Mar. 3, 1900.

**Kidney.**—Chatin and Guinard [6] have in vain endeavored to demonstrate that the kidney forms an internal secretion the injection of which into animals deprived of both kidneys produces any appreciable benefit. [This is what we should expect, and is in accordance with our criticism of the positive results reported by Teissier and Frenkel.[7]]

## NERVOUS SYSTEM.

**Relative Size of Axon and Cell-body of a Neuron.**—H. H. Donaldson [8] states that in the growing (lumbar) spinal ganglia of the white rat the increase in volume of the largest cell-bodies is very closely correlated with the increase in area of the cross-section of the nerve-fibers growing out from them. While the cross-section of the axis-cylinder is, and remains, almost exactly equal to the area of the medullary sheath, the current dictum that the nerve-fibers of larger caliber have the longer course is not true, at any rate for the fibers supplying the thigh of the frog. On the contrary, E. Dunn has shown that the average diameter of the fibers going to the thigh is greater than that of the fibers going to the lower part of the limb.

**The Spinal Ganglia and the Conduction of the Nerve-impulse.**—[It has often been suggested that the afferent impulses in their passage along the posterior spinal roots do not necessarily traverse the cell-bodies of the spinal ganglion cells.] E. Steinach [9] brings forward evidence that the impulse can pass through ganglia in which the vitality of the cell-bodies has been destroyed in various ways, as by artificially produced anemia or by exposure after isolation. He finds that in isolated preparations from the frog the negative variation can be obtained in the posterior root above the ganglion on stimulation of the central stump of the sciatic at a time when the ganglion cells must be supposed, from the results of parallel tests, to be dead; at a time, for instance, when no reflex negative variation can be detected in an anterior root when the sciatic or the corresponding posterior root which has been left in connection with the cord is excited. As Hermann,[1] Bernstein,[2] and Mislawsky [3] have shown, such a reflex negative variation is normally obtainable from a fresh preparation.

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[1] Jour. Physiol., vol. XX, p. 1.

[2] Proc. Physiol. Soc., Mar., 1899 ; Jour. Physiol., vol. XXIV, p. xix ; Ibid., vol. XXV, p. 87.

[3] Proc. Physiol. Soc., Feb., 1899 ; Jour. Physiol., vol. XXIV, p. ix.

[4] Proc. Physiol. Soc., Feb. 13, 1897 ; Ibid., Feb. 12, 1898.

[5] Proc. Roy. Soc., vol. LXV, April 20, 1899.

[6] Arch. de Méd. Exper. et d'Anat. path., tome XII, No. 2, Mar., 1900.

[7] YEAR-BOOK for 1899, p. 964.

[8] Proc. Am. Physiol. Soc.; Am. Jour. Physiol., vol. IV, p. vi.

[9] Pflüger's Arch., Bd. LXXVIII, S. 291.

**Relation of Motor Nerves to the Tissues of the Body.**—J. N. Langley,[4] in an address before the Section of Physiology of the British Association, discusses in a very fresh and interesting style various questions connected with this subject, which has been so brilliantly illustrated by his own researches. His protest against the too common tendency to hasty generalization, nowhere more conspicuous than in this domain, seems to us most timely. We have no right, for instance, to assume that, because some arteries are known to be provided both with vasoconstrictor and vasodilator nerve-fibers, all arteries must be equally well endowed. Especially valuable is his account of the reflexes, or, as he prefers to call them, pseudo-reflexes or axon-reflexes, which can be elicited from the sympathetic nervous system, and which are described more fully in special papers.[5]

**Regeneration of Nerve-fibers.**—J. N. Langley,[6] following up observations previously reported,[7] makes a further contribution to our knowledge of the regeneration of the preganglionic fibers in the sympathetic system. After excision of the superior cervical ganglion, the cervical sympathetic does not recover its function. Accordingly the preganglionic fibers can not form direct functional connection with the peripheral tissues, but can become connected with them only indirectly through the axons of the ganglion cells. [As this observer has previously shown, when the preganglionic fibers are severed without destruction of the sympathetic ganglion, the fibers not only regenerate, but the great majority of them unite with ganglion cells of the same functional group as they were previously united with.] He now concludes that when preganglionic fibers of different lengths are divided,—as, for example, when the lumbar sympathetic chain is severed,—one factor in bringing about this remarkable result is probably the tendency of the several cut fibers to grow to a certain definite length—the same, namely, as they had before.

G. C. Huber [8] demonstrates that after section of the nerves going to the striated muscles the motor nerve-endings degenerate sooner than the sensory. Both may, under suitable conditions, regenerate.

[The question whether nerve-fibers in the central nervous system regenerate has been answered by different writers in different ways, and often in the negative.] Baer, Dawson, and Marshall,[9] after a careful investigation of the phenomena following ligation of the posterior (dorsal) root of the second cervical nerve (in the dog) between the ganglion and the cord, make the statement [which there seems no good reason for doubting] that regeneration of the fibers takes place from the ganglion into the cord, so that the normal reflexes through the respiratory, cardiac, and vasomotor centers may be once more obtained.

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[1] Pflüger's Arch., Bd. LXXX, S. 41; Ibid., Bd. LXXXII, S. 409.

[2] YEAR-BOOK for 1899, p. 968; Pflüger's Arch., Bd. LXXIII, S. 374; Ibid., Bd. LXXIX, S. 423; Ibid., Bd. LXXXI, S. 138.

[3] Centralbl. f. Physiol., Bd. XIV, S. 217.

[4] Lancet, Sept. 23, 1899.

[5] Soc. de Biologie, Jubilee Number, 1899; Richerche di Fisiologia dedicate al Prof. Luciani, 1900, p. 23; Jour. Physiol., vol. XXV, p. 364.

[6] Jour. Physiol., vol. XXV, p. 417.

[7] YEAR-BOOK for 1897, p. 1180.

[8] Am. Jour. Physiol., vol. III, p. 339.

[9] Jour. of Exp. Med., vol. IV, No. 1, 1899.

## SPECIAL SENSES.

**Photography of the Retina.**—Nikolaew and Dogiel [1] have published an interesting preliminary communication on this subject, accompanied by the best specimen of retinal photography we have seen. The amount of detail shown, considering the great technical difficulties, is remarkable.

A. Beck [2] has studied the **retinal currents** in the cephalopod eye (in Eledone moschata). His results differ considerably from those of the classic research of Kühne and Steiner on the retina of mammals and amphibia. When light is allowed to fall on the cephalopod retina, there occurs a strong positive variation, which does not give place to a marked negative variation as in Kühne and Steiner's experiments. Beck, accordingly, is unable to admit that the negative variation is the important thing, as these observers supposed. On the contrary, the real variation of the retina is the positive one, and its seat is the layer of the rods and cones, since in the cephalopod eye the structure called the retina contains only this layer, the other layers of the vertebrate retina being represented in the optic nerve and ganglion.

**Theories of Hearing.**—E. ter Kuile [3] has formulated the theory that when the foot of the stapes is pressed into the oval foramen the displacement of the perilymph in the scala vestibuli causes a downward bulging of the membranous canal of the cochlea, including the basilar membrane, into the scala tympani, since it is only in the direction of the yielding membrane which fills the foramen rotundum that room can be made for the displaced fluid. When the foot of the stapes moves outward, the membranous walls of the scala media bulge up into the scala vestibuli. The effect of these movements is a sheering of the membrana tectoria on the basilar membrane, and consequently a mechanical stimulation of the hair-cells which occupy an intermediate position between them. The bulging begins at the base of the cochlea in the neighborhood of the foramen rotundum, and is propagated upward along the basilar membrane for a distance which varies according to the frequency of oscillation of the foot of the stapes; that is to say, according to the pitch of the note. The higher the note, the smaller is this distance, and therefore the shorter is the tract of hair-cells excited. But for a note of given pitch the distance is always the same. The movements of the liquids of the labyrinth which cause the excitation of the auditory nerve-fibers are massive and not molecular (that is, are not themselves sound-waves).

M. Meyer [4] points out that this theory is the same as that previously published by himself[1] as regards its essential conception, that the stapes at the beginning of its movement causes no other effect than a bulging of the membranous structures between the scala vestibuli and the scala tympani in the neighborhood of the round foramen; and he truly remarks that it is a good sign of the correctness of his fundamental principle that another investigator should have independently arrived at it. But he takes exception in many points to the manner in which ter Kuile works out his theory. He agrees with him in asserting that molecular waves in the perilymph can not possibly play any rôle, since their velocity of propagation is too great in comparison with the frequency of oscillation of the stapes. Both these writers severely criticize the new "sound-picture" theory of J. R. Ewald,[2] according to which stationary waves are produced in the basilar membrane or some portion of it, like the Chladni's figures produced when a metal plate on which sand has been strewed is set vibrating. [All these newer theories necessarily break with the resonator theory of Helmholtz, which, indeed, appears to be out of harmony with certain well-established facts, and, in spite of its splendid services in the past, must probably now be regarded as inadequate. It is as yet too early to make a definite choice between its numerous rivals.]

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[1] Pflüger's Arch., Bd. LXXX, S. 236.

[2] Pflüger's Arch., Bd. LXXVIII, S. 129.

[3] Pflüger's Arch., Bd. LXXIX, S. 146; Ibid., S. 484.

[4] Pflüger's Arch., Bd. LXXXI, S. 61.

## REPRODUCTION.

J. Loeb[3] has made a contribution of far-reaching importance to our knowledge of the significance of fertilization by the discovery that the unfertilized eggs of certain sea-urchins (Arbacia and Strongylocentrotus) can develop in sea-water to which magnesium chlorid has been added in a certain proportion, just as if a spermatozoon had entered the egg. In a further communication[4] he shows that this is not due to a specific action of the magnesium chlorid, since it can be replaced by other salts, and even by substances that are not electrolytes. The essential thing is that the osmotic pressure of the surrounding solution should be increased, and that in consequence the eggs should lose a certain amount of water. The phenomenon has also been obtained with the eggs of the star-fish, and is probably common to the whole Echinoderm group. [Artificial parthenogenesis is, however, not confined to this group. It has been produced by other observers; for instance, by Morgan in the Nemertea and Gephyrea.] And Loeb[5] has recently proved that an increase in the osmotic pressure of the sea-water will also produce the phenomenon in the unfertilized eggs of Chætopterus, a marine annelid. In this case KCl or perhaps K ions seem to possess a specific action, so that if the eggs are put for only 3 minutes into a certain solution containing KCl, they will then develop parthenogenetically in normal sea-water.

In another paper[6] the same writer attempts to answer the question,

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[1] Pflüger's Arch., Bd. LXXVIII, S. 346; Zeit. f. Psychol. u. Physiol. d. Sinnesorg., Bd. XVI, S. 22.

[2] Pflüger's Arch., Bd. LXXVI, S. 147.

[3] Am. Jour. Physiol., vol. III, p. 434; Jour. Am. Assoc., Oct. 28, 1899.

[4] Jour. Physiol., vol. IV, p. 178.

[5] Science, vol. XII, p. 170; Am. Jour. Physiol., vol. IV, p. 423.

[6] Arch. f. Entwickelungsmechanik, Bd. VIII, S. 689.

Why is the regeneration of portions of protoplasm devoid of a nucleus impossible? His solution [and the suggestion is an interesting one as a basis for future work] is that the nucleus contains substances which favor oxidation, and that in its absence the oxidative power of the protoplasm is not great enough for regeneration. The favorable influence of weak alkalies on the development and growth of Arbacia larvæ [1] he attributes to their action in increasing the oxidative processes in the living substance. [That a portion of an ovum of the sea-urchin not containing a nucleus may develop when impregnated by a spermatozoon has been stated by Y. Delage.] He has lately [2] extended this observation to the ova of other animals, and concludes that the essential process in fecundation is the union of the nucleus of the spermatazoon with the cytoplasm of the ovum, and not its fusion with the female nucleus.

[1] Arch. f. Enterickelungsmechanik, Bd. VII, S. 631.

[2] Compt. rend., Oct. 23, 1899; Jour. Am. Med. Assoc., Dec. 2, 1899.

# LEGAL MEDICINE.

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**Epitome.**—During the year 1900 the holding of the First International Congress of Medical Deontology at Paris may be held to some extent to mark an epoch in that part of legal medicine which relates to the status of the physician. The unanimous opinion of those present appeared to be that the inroads made by the various phases of contract practice, on the one hand, and the extension of eclectic and irregular practice, on the other, combined with the keen competition in all ranks of the medical profession, is ominous in its significance. No remedy of general application was, however, formulated by the congress.

As important text-books and monographs which have appeared we would indicate those of Dieckerhoff,[1] on "Veterinary Legal Medicine," of which a second edition was called for within 4 months, and Bailey's American edition of Goliebiewski's "Diseases Due to Accident."[2] The concluding volume of R. Stern's monograph on the same subject has also appeared,[3] and a special year-book on this specialty is announced.[4] A. N. Taylor has rendered a real service by reprinting the series of articles on **the law in its relation to physicians,**[5] published serially in the "New York Medical Journal." An English translation of S. Baudry's well-known little work, **"Injuries to the Eye in Their Medicolegal Aspect,"**[6] is good, though not compendious in its treatment of the subject. The work of O. Amedo on medicolegal dentistry has appeared in German.[7] Further issues of Lesser's valuable stereoscopic medicolegal atlas have also appeared.[8] Krafft-Ebing's work on psychiatric legal medicine has appeared in French,[9] as well as a collection of observations by A. Raffaele[10] and a text-book by C. Emmert.[11]

A. Lacassagne[12] reports on the methods of **medicolegal instruction** in Lyons, and Brouardel reports similarly on those followed in Paris.[13] In consequence of this latter report a previous recommendation of Brouardel's, made in 1884, advising the establishment of a diploma in legal medicine has been finally adopted officially. The details of the course projected are given. [Medicolegal diploma courses on somewhat similar lines have been established in Brussels and in McGill University.] Brouardel, in his report, considers that the course for the diploma should cover postgraduate studies during an entire academic year of 9 months. It should embrace, besides theoretic instruction, (1) practical medicolegal work, under the guidance of medical experts, including, besides autopsies, the examination of wounds and injuries, and assaults, including rape. (2) An elementary practical course on toxicology, in which the candidate shall become familiar with the technic of the ordinary chemical analyses for poisons, without, however, aiming at making him more than an associate to the special chemical expert in reference to future practice. More stress is laid upon his familiarizing himself thoroughly with physiologic experiments to determine the effects of supposed toxic substances, in which he would have to act as principal expert. (3) A thorough study of mental conditions sufficient to enable him to act as principal expert in questions of this nature. The whole course to lead to a written and practical examination. [The course thus outlined is undoubtedly well suited for the conditions which prevail in France. From an American point of view it might be questioned whether the amount of training received in this period really would be sufficient to qualify the candidate to act as an expert on mental questions. It would seem preferable that the training in mental disease should be placed on the same footing as in toxicology, and that at the very least an entire year devoted to psychiatry should be demanded as qualification for a principal expert in that line of work. On the other hand, a moderate amount of training, such as is required in toxicology, would suffice for one who is at most required to act as an associated expert in mental cases. Some experience in teaching in work of this kind leads strongly to the conviction that the highest special training in any one branch of state medicine is hardly feasible as part of a general course in the subject even of one year's duration, and that, while the candidates should all be required to attain a general proficiency before qualifying in their special department, the original course should foresee and provide for the detailed and special study of particular branches. One other point in which the Paris course might be amended is the inclusion of practical training in the civil as well as the criminal side of medicolegal work, as the number of experts required for this far exceeds that needed for the purely criminal branch.[1] The importance of the subject of medicolegal instruction at the present moment can not be overrated.]

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[1] Gerichtliche Thierheilkunde, Schoetz, Berlin, 1900.

[2] Saunders & Co., Philadelphia, 1900.

[3] Fischer, Jena, 1900.

[4] Berlin, 1900.

[5] Appleton, New York, 1900.

[6] F. A. Davis, Philadelphia, 1900.

[7] Felix, Leipzig, 1900.

[8] Barth, Leipzig, 1900.

[9] Ginet, Toulouse, 1900.

[10] Naples, 1900.

[11] Thiem, Leipzig, 1900.

[12] Arch. d'Anthrop. Crim., July, 1900.

[13] Special Report to the Faculty of Medicine of Paris, 1900.

## MEDICAL LAW.

**Medical Responsibility for Injurious Effects of Treatment.**—A medicolegal monograph on the subject has been published by R. Schmidt.[2]

**Property Rights in Medical Case Reports.**—P. Brouardel[3] considers that these may be transferred in the sale of a practice, as the purchaser will become equally bound by the rules of secrecy. [See also under medicolegal decisions.]

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[1] See Phila. Med. Jour., Sept. 8, 1900.

[2] Fischer, Jena, 1900.

[3] Ann. d'Hyg. pub., July, 1900, p. 89.

F. Neugebauer [1] relates 108 cases in which forceps, sponges, swabs, etc., were left in the abdominal cavity after operations. Of 101 cases, 41 ended fatally, 59 healed, and the termination of one was not known. The articles left behind were sponges, 30 times; gauze compresses, 28 times; drainage-tube, 4; Richelot's clamp, 1; artery forceps, 28; signet ring, 1; fragment of broken irrigator, 1; not designated, 17. In 3 cases 2 articles were left. In 38 cases the article was first discovered at the autopsy; in 13 it was voided spontaneously *per rectum*. The inquiry was made in connection with a case in which a surgeon had left behind a pair of artery forceps after laparotomy, was refused permission to reopen the abdomen by the patient's friends, and the forceps caused death by perforation of the iliac artery 6 months later.

H. T. Marshall [2] has written an interesting account of the rise and development of Christian science. The remedies he advocates for this nuisance are: (1) Compulsory reporting to the health department of all births, deaths, and infectious cases attended by disciples of the cult; (2) proper protection of small children against the extravagances of Christian science; (3) general regulation by legislation which shall limit the right to treat the sick to those who have given satisfactory evidence that they possess sufficient knowledge of the conditions of health and disease.

The following are some of the more important judicial decisions in medical cases during the year 1900 in the United States. They have been culled from a large number published from week to week in the medicolegal column of the "Journal of the Americnn Medical Association." The reprinting of the entire series, with a separate index and accurate references to the law reports in which they appear, would be of great value to all who are interested in the judicial aspects of medicolegal work. Nearly 400 abstracts, in all, of these decisions were thus published by the journal of the Association.

The Appellate Court of Indiana was unable to find any precedent for recovery of damages for **mental prostration** due to the **shock of witnessing another person's peril.** [3] In the case in question a mother attributed her condition to fright at a danger to which she saw her daughter exposed. The Court decided not to make a ruling in this sense.

The Supreme Court of New York has decided [4] that the guardian of an infant has a right to waive the privileges regarding **professional secrecy concerning a child.**

The New York civil code, which requires that no revelation of a professional secret can be made without the consent of the patient, has been construed by the Supreme Court of New York not to cover cases where secrecy is invoked to shield a person charged with the murder of the patient.

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[1] Monatsch. f. Geburtsh., XI, 4.

[2] Johns Hopkins Hosp. Bull., June, 1900.

[3] Cleveland, Cincinnati, Chicago, and St. Louis Ry. Co. *vs.* Stewart.

[4] Corey *vs.* Bolton.

The Supreme Court of Ohio[1] rules that "**sound health**" means "freedom from any ailment or disease that seriously affects the general healthfulness of the system and is not a mere indisposition."

The opinion of a witness as to his own **competency as an expert** is held by the Supreme Court of Missouri[2] to be irrelevant and carrying no weight.

The **good-will of a practice,** if sold with it, has been decided by the Supreme Court of Illinois[3] to be included among the obligations of the seller; and if the practice is guaranteed to yield a certain income, and did not do so, the purchaser is entitled to sue in a court of law for breach of contract.

The Supreme Court of North Carolina[4] construes the **contract not to practise** in a certain town nor in the surrounding country holds good during the life of the vendor as regards the town limits, but unless the limits of the outside territory be specifically defined, that this clause of the contract can not be enforced.

The Supreme Court of Indiana[5] holds that a lay **patient is a competent witness** as to whether his rupture is cured or not in case of a suit for contract to cure a rupture.

The Supreme Court of North Carolina[6] holds that a husband can recover the severest damages known to law when a physician is found guilty of having **accelerated his wife's death** by carelessness and inhuman treatment.

The Kentucky Court of Appeals has decided[7] that an **osteopath** is not entitled to charge for his services on the same basis as a physician, but may claim compensation for time employed to the amount to which a laborer or nurse would be entitled.

**Self-treatment of Injuries.**—The Supreme Court of Tennessee has held[8] that the patient's obligation to use all reasonable means to secure proper treatment is sufficiently carried out if for a time, acting without medical advice, he carried out such treatment as a physician would ordinarily have used in treating the injury.

It has been held by the Michigan Supreme Court[9] that **consumption can be properly included among contagious diseases** of which the notification is compulsory, under a statute which covers "other diseases dangerous to the public health"; but a new trial was ordered, as the actual proof was not submitted in the case in question that consumption was a disease of this character.

## DEATH AND DEAD BODIES.

**Cooling of Bodies after Death.**—S. Ottolenghi[10] found that heat was retained longest in the abdomen, next the brain, and then the muscles, which in some cases retained heat longer than the brain.

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[1] Metropolitan Life Ins. Co. *vs.* Howh.

[2] Granley *vs.* Iron Mountain Ry.

[3] Tichnor *vs.* Newman.

[4] Hauser *vs.* Harding.

[5] Wray *vs.* Warner.

[6] Gray *vs.* Little.

[7] Nelson *vs.* State Board of Health.

[8] Arkansas River Packet Co. *vs.* Hobbs.

[9] People *vs.* Shurly.

[10] Ann. d'Hyg. pub., Sept., 1900.

J. Ogier,[1] having tested experimentally the conditions necessary for the **combustion of dead bodies,** finds that a fetus can readily be calcined in a few hours in an ordinary stove. An adult body, even when cut into fragments, requires 40 hours for complete combustion in a stove, and at least 6 hours are required in a blast furnace. Combustion in the open air by means of inflammable liquids is all but impossible. Petroleum, however, gives better results than either alcohol or oil. The phenomena observed under such conditions are minutely described. Human fat when melted is distinguishable without much difficulty from that of most of the larger domestic animals, as its melting-point is higher, except in the case of pork. It is still more easy to distinguish it from vegetable oils. The fats, however, when charred, become much harder to distinguish.

**Observations on the Distribution of Blood in Burned Bodies.**—N. F. Reuter,[2] from the study of 6 personal cases, concludes as follows: (1) Under the influence of heat there is a postmortem displacement of the blood which is still fluid, leading even to extravasation. These changes usually occur in the internal organs, especially the heart and lungs. A diastolic overfilling of the heart chambers with blood can thus occur postmortem. (2) Anemia of the abdominal organs met in burned bodies is probably an agonal condition.

W. H. Welch [3] discusses **conditions due to Bacillus aerogenes** in a most thorough manner. He considers the term gas sepsis the most expressive, and thinks that this is the real cause of most of the cases of alleged uterine air embolism. Particulars of several cases are given. It is pointed out that the uncleanly conditions under which abortionists often work are such as would lead to this mode of infection.

**Death from Starvation.**—H. Dünschmann [4] concludes that starvation brings about an almost complete absorption of the fat in the body except in the case of lipomas. Phosphates and potassium salts preponderate over chlorids and sodium salts in the urine. The sulphates are relatively decreased. It is probable that the hippuric acid excretion is characteristic. Acetone and diacetic acid are so greatly increased as to be valuable for diagnosis. The amount of feces is extremely small and the alimentary canal is reduced in size and contracted. Fat tissue is reduced by 90%; muscle, 30% to 40%; the liver, 50%; and spleen, 60%. The changes in the Nissl granules may be regarded as characteristic. It is not clearly shown that inanition predisposes to tuberculosis. The condition at autopsy shows great reduction in size of the stomach; intestines empty and thin-walled; liver and spleen small; gall-bladder usually distended; and the thymus in children usually almost completely atrophied. Cases of death from starvation are reported by Hartmann.[5] Dünschmann [6] considers that a valuable test for the **diagnosis of starvation** is afforded by the detection of diacetic acid in the urine. The chlorid and iron reaction suffices.

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[1] Thirteenth Internat. Med. Cong., 1900.

[2] Friedreich Bl., Mar., 1900.

[3] Phila. Med. Jour., Aug. 4, 1900.

[4] Viertelj. ger. Med., April, 1900.

[5] Münch. med. Woch., No. 32, 1900.

[6] Münch. med. Woch., No. 39, 1900.

**Putrefaction in Relation to Legal Medicine.**—E. Malvoz[1] has made a most comprehensive study of the subject in all its phases, including the normal bacteriologic flora and putrefactive processes in cadavers, the effect upon the question of respiration in case of fetal lung, the production of toxins, and the effect upon the poisons in the tissues. In every portion of the work the results are based largely on personal observations.

**Hepatic Docimasia.**—A. Lacassagne and E. Martin[2] find that whenever glycogen is present in the liver tissue glucose will be found. They no longer consider the quantitative estimation necessary—small traces may be found in persons who die suddenly in the course of a disease.

A. Lacassagne and E. Martin[3] call attention to the importance of observing the state of digestion in connection with hepatic docimasia.

N. Corin,[4] in studying the **physiology of death by drowning,** found that animals paralyzed by pyridin, strychnin, or chloral died on submersion without the ordinary changes due to respiration under water. He found that decomposition was more rapid in the bodies of persons and animals who died slowly, which he ascribes to the entry of microbes and greater fluidity of blood owing to dilution. Experimentally the time for putrefaction was the same in dogs hanged as in those drowned.

E. Scholz[5] has studied experimentally the **cause of death in burns and scalds.** He finds that, in the case of rabbits, by heating the skin of the ear to a temperature of 70° to 85° C. severe general disturbances, often fatal, resulted; whereas by making the ear anemic and clamping it during the exposure these disturbances did not occur. He infers that the influence of the surface burns is slight in producing the fatal effect as compared with the effect due to the heating of the blood.

Schuchardt[6] reports a case of **sudden death after excision of tonsil.**

A. Haberda,[7] in writing of **epidural hemorrhages in burned bodies,** reports a recent personal case, and also records an observation by H. Hölder[8] in which a precisely similar case was published, having the characteristic fat admixture in the extravasated blood.

Seydel,[9] in a paper upon **death from hemorrhage,** concludes that fatal bleeding does not involve the loss of the whole blood mass, but 1500 gm. to 1800 gm., if lost rapidly, suffice; the loss of blood may cause complications if it interferes with the function of other organs. In fatal puerperal bleeding relatively large quantities are lost, as the bleeding is usually slow.

**Medicolegal Study of Death by Shooting.**—Schaefer[10] contributes a very valuable monopathic article on this subject, unsuitable for abstraction.

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[1] Ann. d'Hyg. pub., Oct. and Nov., 1899.

[2] Ann. d'Hyg. pub., Sept., 1900, p. 273.

[3] Arch. d'Anthrop. Crim., July, 1900.

[4] Ann. d'Hyg. pub., Sept., 1900, p. 263.

[5] Münch. med. Woch., 5, 1900.

[6] Aerzt. Sachv. Ztg., 7, 1900.

[7] Friedreich Bl., Mar., 1900.

[8] Correspondenzbl. d. Würtemb. ärzt Vereines, 1860, p. 241.

[9] Aerzt. Sachv. Ztg., 3, 1900.

[10] Viertelj. ger. Med., Supp., 1900.

C. Geill [1] has made an elaborate statistical study of the results of autopsies upon 586 cases of **rupture of the internal organs** from injury.

## INFANTICIDE.

M. Puppe,[2] in discussing **pulmonary docimasia,** cites experiments on still-born fetuses and animals in which cultures of colon bacillus or earth containing this organism were inserted in the pharynx, and produced an intense development of gas in all the tissues of the body within a few days.

**Influence of Putrefaction on Pulmonary Docimasia.**—Descoust and Bordas [3] strongly affirm that the existence of gaseous putrefaction of the lungs is to be regarded as evidence of respiration.

**Development of Ossific Centers.**—Nobiling [4] bases his studies upon the examination of 2700 fetuses and embryos. He strongly advises measuring the sternum, especially in mutilated bodies. A sternum only 6 cm. long, with only 3 points, will be before term; one 9 to 15 cm. long indicates full term and good development.

Hougonneng [5] has studied the **mineral composition of the human fetus** during the 5 last months of pregnancy in 7 cases. He finds that during this period, apart from the increase in phosphate of lime, corresponding to the bone mass, and in iron, corresponding to the blood mass, the ashes remain of constant mineral composition with 2 exceptions: (1) during the middle period chlorid of sodium increases temporarily; (2) toward the end of gestation calcium is present in excess of what corresponds to the phosphates.

**Infanticide by Strangulation with the Umbilical Cord.**—Dufour [6] reports a personal case where the accused person acknowledged the act, but subsequently recalled her confession and was acquitted by the jury.

## WOUNDS AND INJURIES.

**Concussion of Brain in Medicolegal Relations.**—Niehues [7] devotes a very full article to a general consideration of the subject.

W. Sachs [8] deals with the entire question of **hemorrhage from a medicolegal standpoint,** and gives a very full résumé of the subject, with extensive bibliography, the medicolegal significance of hemorrhages from various regions of the body being very thoroughly gone into.

**Medicolegal Significance of Injuries of the Kidney.**—A. Raude [9] concludes that all injuries of the kidneys are to be classed as severe injuries. Those due to shooting are the most dangerous; next come the subcutaneous ruptures. Stabs and cuts are the least dangerous. The principal dangers are from bleeding and wound infection. Complications are to be regarded as seriously increasing the danger. In many cases they result in permanent invalidism. Subcutaneous injuries may come from direct violence in concussion of the entire body or sometimes from reflex muscular contraction. The chief symptoms are local pain, suggillation, hematuria, and tumor in the region of kidneys. Perirenal injuries are to be differentiated. Flow of urine and blood from the wound occurs in direct injuries. Perirenal bleeding may give rise to floating kidney.

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[1] Viertelj. ger. Med., Oct., 1899, and Jan., 1900.

[2] Thirteenth Internat. Med. Cong., 1900.

[3] Thirteenth Internat. Med. Cong., 1900.

[4] Friedreich Bl., Nov., 1899, ref. p. 57; Deutsche Praxis, 1899, 19.

[5] Acad. de Sci., May 21, 1900.

[6] Ann. d'Hyg. pub., July, 1900, p. 89.

[7] Friedreich Bl., Mar., 1900.

[8] Friedreich Bl., Jan., 1900.

[9] Viertelj. ger. Med., Supp., 1900.

E. Schlesinger [1] deals with the medicolegal significance of **fractures of the base of the skull** in a very full article, developing and indorsing the views of Messerer and Hermann as to the distinction between the effects of compression and expansion, and also as to the effect of the upward pressure of the vertebral column in fracturing the base.

The effects of the fractures upon the optic and auditory nerves are also dealt with, as well as interferences with speech, disfigurement, and intellectual disturbances.

**Indirect Fracture of Larynx.**—P. Wichmann [2] reports a case in which compression of the breast and shoulder from a crushing accident was associated with laceration of the cricoid cartilage and separation from the larynx with rupture of the ligaments.

**The Marks Produced by Pistol Shots.**—G. Hough [3] deals with the question of caliber as influencing the appearance produced at a given range, and gives the results of personal experiments with arms of various calibers.

Thoinot [4] reported on the **marks left by the modern smokeless explosives,** on which he had made numerous experiments. [The full text of his article has not yet appeared.]

**Electric Burns.**—J. M. Elder [5] publishes a series of observations confirming the views of Sharp [6] as to the marked tendency to slow progressive sloughing after electric burns.

C. Walckenaer,[7] in studying **accidents from electricity,** points out that in attempts at rescue the first thing to be done is to cut off the current promptly, and recommends that facilities for this be made more accessible in establishments in which electricity is used, with placards stating the methods of resuscitation to be employed posted prominently, as is done in the life-saving stations. The author does not consider that the theory that direct currents act by direct tissue destruction, and alternating currents by nervous inhibition, is well founded. He concurs in the views of Prévost and Batselli, that brief exposures to alternating currents of 2400 to 4800 volts tended to produce increased contraction of the ventricles at first, while alternating currents of 120 volts produce primary cardiac depression, not aided by artificial respiration, direct currents of 50 to 70 volts producing fibrillar tremor of the heart muscle. The experiments were made with dogs and rabbits.

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[1] Viertelj. ger. Med., Suppl., 1900.

[2] Viertelj. ger. Med., Oct., 1900.

[3] Mass. Medicolegal Soc., Feb., 1900.

[4] Thirteenth Internat. Med. Cong., 1900.

[5] Montreal Med. Jour., Jan., 1900.

[6] Phila. Med. Jour., Jan. 28, 1898.

[7] Ann. d'Hyg. pub., Jan., 1900, p. 88.

**Cause of Death in Electric Shock.**—R. H. Cunningham [1] regards fibrillar contraction of the heart as the determining cause of death in the case of fatal slight shocks from a small amount of current.

Crespin,[2] in discussing the **medicolegal relations of ritual circumcision,** refers to cases of syphilitic and tuberculous infection in the literature.

## ACCIDENT DISEASES.

F. Dreyer [3] has made a statistical study of 200 cases of **accident disease** in the Göttingen University clinic during the period 1884–1898.

A. Benthall [4] has published an interesting series of cases bringing out the disputed medical points in the **assessment of damages** under the new workmen's compensation act.

**Estimation of Disability and Disease Due to Injury.**—W. Johnston [5] gives details and the tables and methods used in estimating temporary and permanent disability by continental government insurance officials.

K. Würz [6] discusses the **traumatic origin of tumors,** giving statistics of 713 cases. Münz [7] reports 4 cases where a traumatic origin of cystadenoma was recognized. Lengneck [8] reports several personal observations of malignant growths following injury. Bruns [9] reports a case of tumor of brain (probably due to trauma). Sandhovel [10] gives a statistical report of 230 cases of trauma and tumors with only 2% of positive results. Würz [11] gives statistics of 714 cases of tumor in which only 2.6% were probably traumatic. The proportion of osteoma was 40% and of sarcoma 7% of the traumatic cases. Uhlemann [12] reports a case of glioma of brain 10 years after trauma. F. Eve and H. R. Davies [13] report a case of myeloid sarcoma of radius following trauma. Keller [14] reports 2 cases of traumatic sarcoma. A. Machol [15] reports the result of a collective investigation on trauma as a cause of tumors, with statistics of 76 cases, full bibliography, and also deals with the subject elsewhere.[16] O. Hahn [17] reports a case of cancer of scalp following injury within a period of 3 months.

**Trauma and Rheumatism.**—Seitz [18] gives brief clinical reports of 38 cases, pruned out of 771, equal to 5%. In these it was monarticular in 8 and polyarticular in 30. The cases occurred among soldiers, the injury being from blows, falls, or sprains. [Other causes do not seem to have been very carefully excluded.] Schulze [19] also reports 2 cases, not very clearly to be shown as anything more than coincidence. Kissinger[1] reports 2 cases of traumatic rheumatism. Maréchaux[2] reports cases of articular rheumatism after trauma. Wolff[3] reports a case of articular rheumatism after injury. L. Becker reports a case of articular rheumatism and trauma. M. Meyer[4] reports a case of peliosis rheumatica after trauma. C. Thiem[5] reports a case of gonorrheal arthritis and injury.

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[1] N. Y. Med. Jour., Oct. 28, 1900.
[2] Ann. d'Hyg. pub., Sept., 1900, p. 262.
[3] Göttingen Thesis, 1899.
[4] Lancet, Mar. 16, 1900.
[5] Montreal Med. Jour., April, 1900.
[6] Beiträge klin. Chir., Bd. XXVI, p. 567.
[7] Centralbl. f. Chir., 25, 1900.
[8] Deut. Zeit. f. Chir., Bd. LII.
[9] Neurol. Centralbl., 11, 1900.
[10] Thesis, Bonn, 1900.
[11] Beiträge f. klin. Chir., Bd. XXVI, p. 567.
[12] Mon. Unfallheilk., 6, 1900.
[13] Lancet, Jan. 20, 1900.
[14] Mon. Unfallheilk., No. 9, 1900.
[15] Beiträge chir. Klin., Strassburg, Singer, 1900.
[16] Thesis, Strassburg, 1900.
[17] Beiträge klin. Chir., XXVI, p. 59.
[18] Mon. Unfallheilk., 1899.
[19] Mon. Unfallheilk., 12, 1899.

Senator[6] discusses the **relation between accident and diabetes.** He had only found trauma in 1% of his cases of diabetes, whereas Cataini found it in 10%. In one case it appeared soon after a laparotomy. He only included trauma occurring immediately before onset of the diabetes. Of the much rarer condition, diabetes insipidus, of which he had notes of 76 cases, 3% or 4% certainly immediately followed trauma, a number of others followed severe psychic disturbances, and one developed 5 years subsequent to a severe injury of the head. Neumann[7] reports 4 cases of diabetes after trauma. In 3 the previous existence of diabetes was not excluded; in the other the injury was a fall, with compound fracture of the leg and sepsis. Lehnhoff[8] discusses the relation between accident and diabetes, and gives a case report.

H. Strauss[9] advocates the **diagnostic use of alimentary glycosuria** for the recognition of traumatic neurosis. The method is that of Naunyn, 100 grams of pure glucose being given in 500 cc. of water on an empty stomach, within a period under 10 minutes. Samples of urine are taken before the test and hourly for 4 hours afterward, and carefully tested for sugar. The author found that while certain conditions, such as fever, thyroidism, alcoholism, lead colic, and a few others, were often associated with alimentary glycosuria, it has been found that glycosuria thus produced is twice as frequent in traumatic as non-traumatic neuroses, so that as a guide the test might be of some value. Strauss found it present in three-eighths of all traumatic neuroses. Haldke,[10] in a study of metatraumatic alimentary glycosuria, examined 25 cases, with 60% of positive results. Neumann[11] tested the frequency of alimentary glycosuria in traumatic neuroses in 30 cases, and found it in 33%.

**Traumatic Heart Diseases.**—Katz[12] discusses the effect of trauma on the heart as a whole; then separately on the muscles, valves, nerves, and pericardium. He considers: (1) That either acute or chronic dilation may follow an accident; (2) that spontaneous rupture of the healthy heart has never been observed, but of diseased hearts more frequently; (3) traumatic myocarditis can be recognized with certainty; (4) chronic traumatic endocarditis certainly occurs and acute traumatic endocarditis probably; (5) motor-sensory nervous disturbances may lead to traumatic cardiac neuroses; (6) the occurrence of traumatic primary pericarditis is relatively frequent. The following data should be established: (1) Was the heart quite healthy previous to the injury? In dubious cases look up records of former insurance examinations. (2) Has the accident made worse an existing heart disease or caused it to become acute? (3) Do the dates of the illness coincide naturally with that of the accident? (4) Which are to be regarded as direct and which as indirect effects?

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[1] Schlesischen Aerzt. Correspondenzbl., 13, 1900; Abstract in Mon. Unfallheilk., 8, 1900.

[2] Aerzt. Sachv. Ztg., 13, 1900.

[3] Aerzt. Sachv. Ztg., 16, 1900.

[4] Aerzt. Sachv. Ztg., 13, 1900.

[5] Mon. Unfallheilk., 1900.

[6] Mon. Unfallheilk., 4, 1900.

[7] Mon. Unfallheilk., 4, 1900.

[8] Aerzt. Sachv. Ztg., 20, 1900.

[9] Mon. Unfallheilk., 12, 1899.

[10] Deut. med. Woch., 1900, 131.

[11] Mon. Unfallheilk., 8, 1900.

[12] Münch. med. Woch., 51, 1899.

Castiaux and Laugier [1] conclude that **valvular lesions from contusions of the thorax** may be caused in healthy persons, and still more readily in persons with heart disease. The aortic valve is most often affected. The auscultatory signs may be those of spontaneous endocarditis or more intense and musical, especially with ruptured valves. The prognosis is worse than in spontaneous valve lesions.

P. Brouardel [2] states that **traumatic endocarditis** is recognizable by dating lesion. This may be helped by sphygmographic tracing, which is irregular during the evolution of the disease, but becomes regular again on cicatrization.

F. Leppmann [3] discusses the question, When should rupture of arterial trunks be regarded as the result of an accident? and reports a case of rupture of the aorta, with very full bibliography.

**Rupture of Lung after Contusion of the Chest.**—G. Bogdan [4] reports a case where in a death 4 hours after contusion of abdomen and chest, without fractures, the lung was found lacerated and the thoracic cavity largely filled with blood.

Garcin [5] reports a case of **rupture of the aorta** 6 weeks after injury.

Freyer [6] reports the following case of **accidental rupture of the aorta**: A man of 38 felt sudden pain in bending over at work and called out that something had burst inside of him. During the next 3 days he felt well and showed no objective symptoms. On the fourth day he suddenly complained of pain and died immediately afterward. The autopsy showed just above orifice a transverse slit in the aortic intima, which was much sclerosed. This opened into the pericardium.

**Disease of the Vessels Following Accident.**—Edel [7] reports a case where symptoms of arteriosclerosis suddenly made their appearance after an accident due to the effects of an explosion.

Wiedemann [8] reports a case of **pulsating exophthalmos after head injury.**

Daulnoy [9] reports a case of traumatic exophthalmos.

Senator [10] records 2 cases of **traumatic pleurisy.**

R. Stern [11] discusses the etiology and course of **traumatic conditions of the stomach mucosa.** He considers that we can recognize clinically (1) a group of acute cases beginning with shock and hemorrhage, and usually ending in rapid healing; these are due to simple lesions of the mucosa. (2) Cases running a chronic course and taking on the character of peptic ulcer with dyspepsia, gastric neuralgia, and repeated hemorrhages.

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[1] Thirteenth Internat. Med. Cong., 1900.

[2] Arch. d'Anthrop. Crim., p. 505, 1900.

[3] Aerzt. Sachv. Ztg., 14, 1900.

[4] Soc. de méd. de Jassy, 1900.

[5] Aerzt. Sachv. Ztg., 16, 1900.

[6] Mon. Unfallheilk., 5, 1900; Berl. klin. Woch., 15, 1900.

[7] Aerzt. Sachv. Ztg., April 15, 1900.

[8] Deut. med. Woch., 9, 1900.

[9] Ophthal. Clinic, Sept. 8.

[10] Mon. Unfallheilk., 2, p. 60, 1900.

[11] Deut. med. Woch., 38, 1899.

F. Jessen [1] reports a case of traumatic gastric ulcer.

**Traumatic Perityphlitis.**—Fürbringer [2] considers that numerous forms of accident or overexertion may cause perityphlitis or act as the principal factor in setting it up. The trauma may act either by lighting up an existing latent appendicitis or causing perforatión, or even cause it to occur in a healthy and empty appendix. Wolff [3] reports a case of traumatic perityphlitis where a blow with the hand on the right side of the abdomen was followed by typical symptoms. Previously had nothing abnormal on palpation of abdomen. H. Schottmüller [4] reports 3 cases of traumatic appendicitis.

**Trauma and Peritoneal Fat-necrosis.**—Simmonds [5] reports a case associated with gangrene of the pancreas following a contusion of the abdomen 5 weeks previously.

Van Engelen [6] reports a case of traumatic cyst of the mesentery. Case report.

Herdtmann [7] reports a case of traumatic femoral hernia from an abscess in the femoral ring.

**Condition of the Urine after Palpation of Kidneys.**—C. Menge,[8] in 21 cases, found that 15 showed albumin and blood-cells subsequent to the examination.

**Traumatic Nephritis.**—R. Stern's [9] conclusions are: (1) Cases of trauma occur in which the symptoms may resemble at first acute nephritis, but autopsy may show no nephritis but extensive necrosis. The course, unless the injury is otherwise severe, is usually benign, the nephritis being probably circumscribed. (2) In rare cases albuminuria may persist for a year or more without other evidences of nephritis. We have no anatomic records of this condition. (3) In a number of cases typical clinical history of chronic progressive nephritis has followed injury. In such cases renal lesions probably preexisted.

C. Thiem [10] reports a case of **exacerbation and uterine prolapse** from injury.

**Traumatic Bubo.**—Rubinstein [11] reports a case in which a sudden pain in the right inguinal region was experienced immediately after heavy lifting, and a bubo developed there. The man had had gonorrhea 2 years before, which had apparently been quite cured. Lissauer [12] reports a case of **osteomalacia after trauma** in a man following sprain of both feet.

**Traumatic Myositis Ossificans.**—C. Rammstet [13] gives the record of a personal case and citation of 15 others.

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[1] Mon. Unfallheilk., 8, 1900.
[2] Aerzt. Sachv. Ztg., 9, 1900.
[3] Aerzt. Sachv. Ztg., 11, 1900.
[4] Mitth. aus. d. Grenzgebiet, 1900.
[5] Münch. med. Woch., 16, 1900; Hamburg Med. Soc., 20, Feb., 1900.
[6] Jour. Med. de Brux., June, 1900.
[7] Mon. Unfallheilk., 6, 1900.
[8] Münch. med. Woch., 23, 1900.
[9] Mon. Unfallheilk., 11, 1899.
[10] Mon. Unfallheilk., 6, 1900.
[11] Arch. f. Unfallheilk., III, 2.
[12] Mon. Unfallheilk., 7, 1900.
[13] Arch. klin. Chir., XXI, 1.

**Estimation of Disability Due to Sciatica.**—H. Ehret[1] gives a description of the special relations of the condition to various common movements, and lays special stress on diagnostic points. The patients sit on the front edge of the chair with the body leaned forward and the knee down. In sitting on the ground the thigh is as much flattened to the ground as possible, knee straight. In changing to a lying position the thigh is somewhat raised. In rising from the chair the patient leans back or brings the body erect to the front of the chair and carries the feet back. In rising from the ground the body is leaned back and raised by both hands. In bending forward the affected leg and foot are carried backward. In walking the foot is turned outward.

Cases of diffuse **muscular atrophy from slight injury** of terminal regions of the extremities are reported by G. Ballet and H. Bernard.[2]

Herdtmann[3] reports 2 cases of **traumatic paralysis agitans.**

Erb[4] reports 2 cases of chronic anterior **poliomyelitis after trauma,** with literary study.

Brodmann[5] reports a case of **traumatic ascending neuritis** and cites two others in the literature.

**Medicolegal Relations of Myelitis.**—An elaborate study of this subject has been made by R. Richter.[6]

Baruch[7] concludes that there exists an altered galvanic resistance of the head in **traumatic neurosis,** as shown by a diminution of the average of electric resistance, but that the range of variation in normal individuals overlaps this to some extent.

The connection between injuries of the eye and fatal meningitis is discussed by M. Wolff,[8] who reports a case in which enucleation was done 17 months after an injury, and was followed by suppuration and infection.

## MEDICOLEGAL TESTS.

**Recognition of Blood-cells in Stains.**—Moser[9] recommends the use of 10% formalin, 10% potassium acetate, and 20% glycerin (Kaiserling fluid) as a medium for preserving the outline and grouping of the blood-cells. It may be lightly tinged with eosin to make the erythrocytes more distinct; and if the supposed blood is in thick crust, sections may be cut.

**Spectroscopic Analysis of Blood.**—C. Ipsen[10] recommends for the extraction of old blood stains or charred blood the use of 5% to 10% chemically pure acetate of potassium in pure absolute alcohol. It is to be digested at 38° to 40° C., and gives the spectrum of alkaline hematin, readily converted by sulphid of ammonium into that of reduced hematin.

**Value of the Hematoporphyrin Test.**—C. Ipsen[11] has tested the

[1] Mon. Unfallheilk., 2, 1900.

[2] Arch. gén. de méd., May, 1900.

[3] Mon. Unfallheilk., 6, 1900.

[4] Deut. Zeit. f. Nervenh., XI.

[5] Münch. med. Woch., 24 and 25, 1900.

[6] Friedreich Bl., Oct., 1899.

[7] Thesis, Breslau, 1900.

[8] Aerzt Sachv. Ztg., 13, 1900.

[9] Viertelj. ger. Med., Oct., 1900.

[10] Viertelj. ger. Med., Jan., 1900.

[11] Viertelj. ger. Med., July, 1900.

extent to which the blood normally present in the various organs and tissues will give a positive hematoporphyrin reaction in the case of charring of these organs. It was found that the lung, liver, spleen, kidney, heart and skeletal muscle, brain, and placental tissue gave the reaction (except in one case of severe anemia). Skin and subcutaneous tissue gave a negative result unless the seat of blood extravasation due to traumatic [or mechanical] causes.

M. Richter [1] emphasizes the following sources of difficulty in obtaining **hemin crystals**: (1) Methemoglobin is not readily oxidized to hematin. (2) In old stains there is danger of a premature reduction to hemochromogen during the period of solution; hence if the test is made early, it may be negative. In doubtful cases a contact of 24 hours with strong acetic acid may be needed to insure the necessary hematin formation. (3) The preparation should not be heated above 120° C. and a too rapid evaporation of the glacial acetic acid should be avoided.

E. L. Walker [2] considers that for the medicolegal **differentiation of human and animal blood** the study of the leukocytic elements by differential staining methods is of value. He advises dissolving a fragment of the stain in distilled water and testing first with Ehrlich's tri-acid stain. "If leukocytes containing minute granules are present, whether alone or associated with leukocytes containing large granules, a second preparation is to be stained with eosin glycerin. If the small granules stain, it is not human blood. If no small granules are seen stained with the glycerin-eosin, re-stain the specimen with tri-acid to make sure of their presence in this preparation. If leukocytes containing small granules are then seen, the blood is that of man or an ape."

**Examination of Seminal Stains.**—Dvornitschenko [3] claims to have met seminal stains in which, while numerous spermatozoa could be recognized microscopically, the test for Florence's crystals proved negative. The conditions under which this occurred are not definitely stated.

**Florence Reaction.**—Davydoff [4] finds that the characteristic crystals can be preserved for several months in sealed tubes. He has been able to obtain a similar reaction from the genital organs of certain plants (hyacinth, chrysanthemum) with the development of a spermatic odor.

**Human Nails.**—P. Minakow,[5] from the examination of the hands of 278 persons, finds that: (1) The nails are broadest on the hand most used, and of equal breadth in the ambidextrous. (2) The difference in width of corresponding nails averages from ¼ mm. to 2 mm., and rarely exceeds the latter. (3) The sum of the width of the nails on the hand most used may exceptionally be less than that of the opposite side owing to the effects of disease or injury. (4) The thumbnail is usually widest, the order being as follows (1, 3, 4, 2, 5): middle finger, ring-finger, forefinger, little finger. (5) The nails are flatter on the hand used most;

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[1] Viertelj. ger. Med., July, 1900.

[2] Boston Jour. Med. Sci., June, 1900.

[3] Viertelj. ger. Med., July, 1900.

[4] Vratch, No. 16, 1900; abst. in Arch. d'Anthrop. Crim., July, 1900, p. 429.

[5] Viertelj. ger. Med., Oct., 1900.

the flattest nails are those of thumb and forefinger; the least flat, those of ring-finger and little finger. Persistent pressure by finger-tips or frequent paring tends to flatten the nails. (6) The thickness of the nails diminishes from the thumb as follows: 60; 51; 46; 42; 41. (7) The width of the nails corresponds closely to the width of the chest.

J. Moeller [1] has published a series of valuable microscopic studies of the various forms of **animal hairs,** illustrated by 140 woodcuts.

O. Mönkemuller and L. Kaplan [2] recommend for taking **records of footprints** that the person to be examined should wear socks moistened with 10% alcoholic chlorid of iron solution. The paper walked on is afterward treated with sulphocyanid of ammonium.

A. Praut [3] gives a review of the subject of identifying **footprints and handmarks.** He recommends especially the plan of taking the imprint on paper exposed to iodin vapor, obtained by heating iodin crystals in a beaker, and subsequent treatment with starch as a means of restoring imprints which have been erased.

Gross [4] recommends **for taking impressions and making casts** the use of 1 part cement, 2 parts chalk, $\frac{1}{4}$ part strong size, and $\frac{1}{8}$ part petroleum. This makes a mass which is soft and nonporous and which can be molded as well as clay and sets as hard as baked clay. For the preservation of plaster-of-Paris models it is recommended that they shall be dried as thoroughly as possible as soon as the plaster sets. This does away with the friability which is the great drawback of plaster-of-Paris.

For **taking copies of documents** Gross [5] recommends printing direct on sensitized paper through the manuscript in strong sunlight. This gives a copy resembling the original more than a photograph will. Gross [6] also recommends for preservation of handwriting when in poor condition the use of varnishes.

Gross [7] recommends the use of a varnish consisting of 1 part stearin and 2 parts collodion for **preserving sketches,** as after this the surface can be washed if desired.

Levinson [8] advocates the employment of **radiography as a means of identification,** taking exact measurement of the bones. It has, however, admittedly, the main drawback of the Berthillon method: viz., that it can be used only in the case of adults. Corrections are given with the view to enabling the error in perspective to be calculated.

J. W. White [9] gives the report of the X-ray Committee of the Association of American Surgeons. The conclusions are as follows: (1) The use of the Röntgen rays is not necessary in all cases of fracture. (2) In fractures of the spine and base of the skull the results at present are not satisfactory. (3) The proper use of the skiagraph calls for an expert surgical interpreter of the conditions shown. (4) The results in non-union and good callus formation may not be appreciable by the skiagraph in recent cases. (5) The exact causes of x-ray burns are not known; their prevention is not difficult. (6) In the detection of foreign bodies it is of the greatest possible value. (7) The legal status of the skiagraph as evidence differs in various States. (8) The technical work may be done by a special expert, but the interpreter requires to be a general surgeon [and a good anatomist].

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[1] Arch. krim. Anthrop., Nov., 1899.

[2] Arch. f. Anthrop., Dec. 27, 1899.

[3] Neurol. Centralbl., No. 17, 1900.

[4] Arch. krim. Anthrop., Mar., 1900.

[5] Arch. krim. Anthrop., May 17, 1900.

[6] Arch. krim. Anthrop., May 17, 1900.

[7] Arch. krim. Anthrop., Mar., 1900.

[8] Arch. krim. Anthrop., Nov., 1899.

[9] Am. Jour. Med. Sci., July, 1900.

## TOXICOLOGY.

D. Scherbatscheff[1] has studied experimentally the **duration of elimination of arsenic,** and concludes that the same quantity may disappear at variable rates in different animals. It is more rapidly excreted by rabbits than by dogs, and more slowly by large than by small animals. The elimination is more prolonged in larger doses, but not in direct proportion. The results on animals can not be directly applied to man. The latest period of detection in man was 70 days, whereas it could be recognized after 120 days in rabbits and 160 days in dogs.

Gauthier[2] has studied the **normal presence of arsenic** in animals and its distribution in the various organs, and finds minute traces to be normally present in the thyroid, thymus, and brain, obtaining 1 mg. from 127 gm. of tissue. He describes his methods for the detection and estimation of minute traces of arsenic in the organs. The essential point is the use of successive changes of nitric and sulphuric acids for destroying the organic matter, as he found that by the nascent chlorin method part of the arsenic escapes as a volatile chlorid.

Chas. Harrington[3] points out the diagnostic difference between **food-poisoning and that by metallic irritants.**

**Toxic Accidents Resulting from Use of Chemical Preservatives in Food.**—Besides the abundant evidence adduced in this country, P. Brouardel and G. Pouchet[4] pronounced strongly against the addition of such substances as boric acid, formol, or salicylic acid as preservatives, and were indorsed by a resolution of the Congress.

**Poisoning by Balloon-gas.**—Crone[5] reports a case of multiple accident from this cause in which the victims, after recovering from the initial cyanosis, developed symptoms of arsenical poisoning from the arseniureted hydrogen present.

**Poisoning of Aeronauts by Arsenide of Hydrogen.**—Durand[6] has collected the literature of fatal accidents occurring in connection with the deflation of balloons. He considers that persons employed in this work should use protective masks.

W. Sachs[7] has published a monograph on **carbonic oxid poisoning.** A. Bloch[8] reports a case of hysteric [?] aphasia induced by carbon monoxid poisoning.

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[1] Viertelj. ger. Med., April, 1900.

[2] Compt. rend. de la Soc. de Biol., 1899, No. 23, p. 929.

[3] Mass. Medicolegal Soc., Oct. 1, 1899.

[4] Thirteenth Internat. Med. Cong., 1900.

[5] D. Milit. aerztl. Ztg., 3, 1900.

[6] Ann. d'Hyg. pub., July, 1900.

[7] F. Wieweg, Brunswick, 236 pp.

[8] Münch. med. Woch., 28, 1900.

**Chronic Lead-poisoning and Accident.**—F. Bähr[1] reports a case remarkable owing to the sudden appearance of symptoms after an injury.

R. Stern[2] reports a case of poisoning by external use of naphthol.

A case of fatal carbolic acid poisoning from skin absorption is recorded by R. Abrahams.[3]

Poisoning by **anilin** from dye-stuff used for coloring leather by absorption through the skin has been observed. G. Brouardel[4] reports a series of 10 children who showed acute symptoms of anilin-poisoning shortly after putting on boots recently dyed with anilin black. The dye was found to contain 90% of anilin oil.

**Benzol and Toluol Poisoning.**—A. Friedländer[5] gives reports of 4 cases. He finds that alcoholism acts injuriously, and may determine the sudden development of symptoms in case of latent poisoning.

Brouardel, Ogier, and Vibert[6] report a case of **atropin-poisoning** where the drug was administered at frequent intervals with criminal intent, the patient presenting the classic symptoms, and repeatedly undergoing relapse. In all, he was partly unconscious for 9 days. His wife, who was suspected, was found to have bought atropin solutions at 4 different pharmacies; in all, 9 lots—making a total of 0.9 centigrams. She was condemned to hard labor.

**Phenylhydrazin-poisoning.**—A case is reported by Carminer,[7] who considers that it produces a definite type of anemia.

Microscopic examinations of the **optic nerve** in **male-fern poisoning** made by Y. Okamoto[8] show that the optic nerves of dogs poisoned with male-fern show well-marked degenerative changes in the nerve element, followed by hyperplasia of the nerve sheath.

## QUESTIONS RELATING TO SEX.

Wichmann[9] studied under Fürbringer a case of **fatal injury from coitus.** The coitus took place in the standing posture during the ninth month of pregnancy in a person of 26. Profuse bleeding ensued, which was found to come from the right side of the clitoris. A good review of the literature of the subject is given, with statistics of 170 coitus injuries, of which 21 were fatal. The predisposing causes, apart from brutal violence or drunkenness, are excessive size of the penis, disproportionate age of the female, pathologic condition of the genitals, malformations, puerperal or pregnant conditions, and standing position during coitus. Wichmann has collected 20 cases of fatal violence from coitus. Death occurred in 13 cases from hemorrhage and in 7 from septic infection. The tissues injured were: Extension of vagina, 4 times; posterior vaginal wall, 4; left side of vagina, 1; right side of vagina, 1; hymen, 1; clitoris and urethra, 2; varix of clitoris, 1;

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[1] Aerzt. Sachv. Ztg., No. 20, 1900.

[2] Therap. Monatsh., Mar., 1900.

[3] Pediatrics, Mar. 15, 1900.

[4] Ann. d'Hyg. pub., Aug., 1900.

[5] Neurol. Centralbl., 4, 1900.

[6] Ann. d'Hyg. pub., Jan., 1900.

[7] Berl. Soc. innerer Med., May 21, 1900.

[8] Viertelj. ger. Med., Jan., 1900.

[9] Aertz. Sachv. Ztg., 4, 1900.

wound of peritoneum, 2. In 4 cases the victim was under 12 years of age. Two cases were of rape; 5 occurred during the wedding night. Pregnancy existed in one case and hemophilia in one.

R. Braun [1] has studied **perforation of the uterus during gynecologic operations.** The commonest cause is instrumental separation of the placenta. Perforation of the wall by dilators is rare, and by sounds rarer still. With the clamp forceps not only has the uterus been perforated, but even coils of intestine dragged down. The puerperal uterus, especially if septic, is readily perforated. Further predisposing causes are tumors, especially malignant moles, atrophy, and edematous states.

L. Lewin and M. Breuning [2] have published a **monograph on abortion.**

**Sulphuric Acid as an Abortifacient.**—S. Egger [3] reports the case of a girl who died from the effects of a half-cupful of 30% sulphuric acid given to produce abortion. The symptoms of irritant poisoning developed immediately, and were followed by profound emaciation and debility. She was delivered of a dead fetus 2 months later, and died at the end of the third month. The stomach showed extensive cicatrization at the pylorus.

**Medicolegal Relations of Marriage.**—P. Brouardel [4] has issued a further addition to the series of works by him, dealing with hysteria, impotence, sterility, pregnancy, and other sexual medicolegal problems arising in connection with married life. Like the other volumes of the series, it is enriched by a series of illustrative cases. Several of the chapters in the book have been published separately in the "Annales d'Hygiene Publique."[5]

Nina Rodriguez [6] has given a careful study to the various **injuries of the hymen,** and is of the opinion that in the majority of instances the condition of the hymen does not permit of an absolutely safe diagnosis.

A. Haberda [7] finds that **anatomic evidence of rape** is often wanting, in spite of the fact that coitus has been completed. The hymen varies greatly in form, elasticity, and size of lumen, and may present appearances liable to lead to confusion. He considers that to be fully characteristic the laceration must extend through the entire hymen and involve, at least slightly, the vaginal wall.

**Vicarious Hemorrhages.**—E. Schaeffer [8] reports a case where a woman of 20 was the victim of a violent assault with attempt at rape. The same evening she began to cough and to spit blood, and this continued for 5 days. This was repeated at intervals of a month for 3 successive months. The apparent cause was the fright received.

Calle [9] in speaking of medicolegal relations of **forced labor and cesarean section,** insists on the uselessness of postmortem cesarean section and lays stress upon the advantages of manual extraction. The cervix of a pregnant woman, he states, is always readily dilated and entered during the agonal period or immediately after death, whether labor has begun or not.

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[1] Wien. med. Presse, 6, 1900.

[2] Hirschwald, Berlin, 1899.

[3] Freidreich Bl., No. 4, 1900.

[4] Le Mariage, Baillieu & Co., Paris, 1900.

[5] Nov., 1899; Dec., 1899; Jan., 1900.

[6] Ann. d'Hyg. pub., June, 1900.

[7] Monatsch. f. Geburtsh., Bd. XI.

[8] Viertelj. ger. Med., Jan., 1900.

[9] Bull. de la Soc. de Méd., Nov. 5, 1900.

## MENTAL QUESTIONS.

Dupré and Rocher[1] consider that **criminality resulting from hypnotic suggestion** can easily result in the case of minor offenses and in suitable subjects, but that any departure calculated to shock the moral sense of the individual is unlikely to occur.

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[1] Thirteenth Internat. Med. Cong., 1900.

# PUBLIC HYGIENE AND PREVENTIVE MEDICINE.

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## THE MANAGEMENT AND CONTROL OF INFECTIOUS DISEASES.

**Compulsory Notification of Measles and Infectious Pneumonia.**—Vallin [1] discusses the propriety of notification of measles and infectious pneumonia. The following figures show the deaths from measles and from scarlet fever, and the number of disinfections practised in each:

| SCARLET FEVER. | 1895. | 1896. | 1897. | 1898. | 1899. |
|---|---|---|---|---|---|
| Deaths . . . . . . | 178 | 170 | 65 | 117 | 195 |
| Disinfections . . . . | 8,336 | 8,914 | 4,877 | 11,355 | 11,448 |
| MEASLES. | | | | | |
| Deaths . . . . . . . | 679 | 658 | 821 | 865 | 904 |
| Disinfections . . . . | 2,633 | 2,535 | 2,955 | 3,090 | 2,232 |

The total number of disinfections made by the municipal service has increased from 38,000 in 1895 to 64,000 in 1899, and yet the practice of disinfection after measles appears to have diminished in the same time, comparing the results of 1899 with those of 1895. At the advice of Vallin and others the Academy of Medicine adopted a proposal on Feb. 27, 1900, to include measles among the notifiable diseases, under the law of 1892. In military practice the following method of dealing with measles is carried out: The first case is sent to hospital; the sick room and its contents are disinfected the same day; unless lack of space in the barracks prevents, the room is vacated, the floors are washed with cresyl, and the walls are lime-washed or sprayed with an antiseptic. Every morning for a week the bedclothes are carried into the yard and exposed to the open air and sunlight. For at least a fortnight the men who had occupied the same room and come into contact with the patient have to report themselves to the doctor, who examines the skin, mucous membranes, palate, uvula, etc.; every one who comes up to sick call for any illness whatever is subjected to a special examination in view of measles. In this way faint rashes are often detected on men who did not suspect that they could infect their comrades by walking with them. Prompt diagnosis and isolation lessen at least by one-half the risks of contagion and spread of disease. After prolonged discussion, the Academy, at the suggestion of Graucher and Vallin, also adopted the following proposition: "The Academy is of the opinion that infectious pneumonia and infectious bronchopneumonia should be included in the list of compulsorily notifiable diseases." This discussion also brought out some facts with regard to the cost of disinfection in Paris, the charges varying from nothing for dwellings of low rental to a maximum of 100 francs for those of the highest class. The charge for a house renting for about 6000 francs, and for disinfecting all rooms and removal and disinfection of their contents, is only 45 francs. The city of Paris spends annually 600,000 francs for this service, and receives back only 40,000 francs as charges, so that the service is practically gratuitous. Several private concerns have been started to carry on disinfection; some do the work in a shiftless and expensive manner.

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[1] Rev. d'Hyg., April, 1900.

**Diffusion of the Plague.**[1]—The following summary shows briefly that from 1879 to 1898 not a single year has passed without the development of plague in at least one country, and in later years the disease has been present in several countries at one and the same time: In 1880 plague was reported to be present in Mesopotamia; in 1881 it was present in Mesopotamia, Persia, and China; in 1882, in Persia and in China; in 1883, in China; in 1884, in China and in India; in 1885, in Persia; in 1886, in India; in 1887, in India; in 1888, in India; in 1889, in Arabia, Persia, and China; in 1890, in Arabia, Persia, and China; in 1891, in Arabia, China, and India; in 1892, in Mesopotamia, Persia, China, Russia, and Tripoli; in 1893, in Arabia, China, Russia, and India; in 1894, in Arabia, China, and India; in 1895, in Arabia and China; in 1896, in Arabia, Asia Minor, China, Japan, Russia, and India (also 2 suspected cases in the London docks); in 1897, in Arabia, China, Japan, India, Russia, and East Africa; in 1898, in Arabia, Persia, China, Japan, India, Russia, East Africa, and Madagascar (also 3 fatal cases in Vienna). It must also be remembered that since 1890, and probably long before that time, plague has been occurring in Central Africa, whence it might have been carried northward by Arab travelers to Egypt and Tripoli in past years, or eastward toward the ports in communication with Jeddah and the Red Sea coast.

**Destruction of Rats and Mice for Prevention of the Plague.**—Doriga[2] contributes a paper on this subject. He says: "The French Government has charged the Comité Consultatif d'Hygiene with the investigation of this question. Certain navigation companies have also taken measures for the destruction of rats and mice upon their vessels. The Compagnie des Messageries Maritimes offered a bounty to their sailors for every rat caught on board. Instructions have been adopted by the Minister of the Interior. These instructions urge the importance of preventing the access of rats to floating hospitals and ships at the wharves, at sea, and on arrival in port. If the dead bodies of rats are found on board, a bacteriologic examination must be made; and if the plague bacillus is found, the ship must be discharged, the cargo and passengers' and crew's baggage disinfected, the vessel fumigated throughout and the dead rats burned." See also articles by Mereshowsky,[1] who studied the question of infecting rats and mice with destructive bacilli other than those of plague, with the purpose of exterminating them. Abel [2] also has experimented in the same direction. It was found necessary to discriminate between wild species and those which live habitually in contact with man. The field-mouse, for example, appears to be endowed with slight susceptibility to infection.

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[1] Twenty-eighth Ann. Rep. Local Gov. Board of Eng., p. 202.

[2] Rev. d'Hyg., Aug., 1899.

**Plague Precautions in English Ports.**—The Chief Medical Officer of the Local Government Board of England [3] briefly details the measures adopted in England as follows: "The system adopted in this country in such cases is the medical examination of all persons on board; the removal to hospital of any person either suffering from plague or suspected to be so suffering; the disinfection of articles believed to have had the opportunity of becoming infected, and of those portions of the vessel occupied by the sick; the registering on board of the names and addresses of all the remaining persons, including the crew, such persons being then free to leave the ship and go to the addresses given; and, lastly, the transmission to the sanitary authorities of the names and places of residence of persons leaving the vessel for their respective districts with a view to such persons being maintained under supervision of the medical officer of health during the 10 days which have been determined on as representing, so far as administrative purposes are concerned, the period of incubation of the plague. The system embodied in these measures is that which England has now for a long period adopted with regard to exotic diseases; it aims at arresting at our ports actual cases of foreign disease, plague, cholera, and yellow fever, and of securing the disinfection of articles which may reasonably be held to have incurred risk of infection. For the rest, it imposes no restrictions on either individuals or articles imported; but it relies on the internal sanitary administration of the country to control at the onset any infection which may perchance evade the precautions adopted at our ports. Thus far it has been singularly successful, as regards the 3 diseases to which it has been applied; and the responsibility which devolves under it on local sanitary authorities, to organize their public health departments so that they shall be always prepared effectually to deal with any chance infection which may reach them from abroad, has had the inestimable advantage of helping to secure at the same time a standard of health at home which has resulted in an immense saving of life."

With regard to the possibility of the **spread of the plague by means of merchandise,** he makes the following statements: [4] "Having regard to our present knowledge concerning the etiology of bubonic plague, I feel justified in holding the view that, in so far as danger of the introduction of that disease into this country is concerned, the first and most important point to be held in view in our port sanitary administration is such medical inspection of persons arriving from infected countries as shall go to secure the detection and immediate isolation of those mild attacks which are mainly identified with slight or indolent bubonic enlargements and indications of general malaise. Next to this, and always to be observed, is the disinfection of all articles of clothing, etc., which have had opportunity of becoming infected either before they were packed for the journey or since. Thirdly, comes the disinfection of those portions of the vessel which may have been used by patients suffering from or suspected to have had the plague. And, lastly, measures should be adopted to prevent the conveyance of the disease to the shore from an infected ship by means of rats, which are peculiarly susceptible to the plague infection. But no system of inspection or other restriction is perfect; and hence for the rest we must trust to such local sanitary administration of our towns, villages, and hamlets as will deprive a disease such as plague of the means of diffusing itself, should a chance case make its way inland."

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[1] Centralbl. f. Bakt., XVII, p. 742, and XX, pp. 85 and 176.

[2] Centralbl. f. Bakt., XXI, p. 497.

[3] Twenty-eighth Ann. Rep., Suppl., 1899, p. XXX.

[4] Twenty-eighth Ann. Rep. Local Gov. Board of Eng., Suppl., p. XXXVII.

**Protection of Railway Employees from Malaria.**—Baldi [1] presents a statement of the measures taken by the Society for the Study of Malaria, under the direction of Celli, upon the railway between the stations of Prenestina and Salone, near Rome, in an intensely malarious region, for the protection of railway employees. The methods of prevention are detailed under these heads: (1) Protection of the dwellings of laborers. This was done by means of screens at the windows and doors, which proved successful in securing immunity of the women and children in the houses, but not of the heads of families who performed night service out of doors. (2) Personal protection by means of veils, gloves, and the use of special soaps intended to prevent mosquito bites. These measures were not successful. (3) Destruction of mosquitos by burning at night at the railway stations a mixture called zanzoline, made of chrysanthemum flowers, valerian root, and other substances, by means of which sleep at the railway stations became possible when the mixture was in use. The results, on the whole, were very encouraging.

**Malaria in Rome.**—Santori [2] shows by means of a series of illustrative charts the relation of rainfall to the prevalence of malarial fever: (1) Two distinct periods—an endemic period, from January to the first 10 days of July, and an epidemic period for the remainder of the year until December. (2) In the endemic period the disease is mild, the cases are few, and its course uniform or regular. (3) An epidemic in the spring is almost unknown. (4) The epidemic period begins suddenly after the first 10-day period of July. (5) The severity of the epidemic may have relation to the same factors. (6) The rainfall of

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[1] Supplemente al Policlinico, Feb. 24, 1900, p. 536.

[2] La Malaria nella Provincia di Roma (1888–97) sua ripartizione nei Communi e suoi Rapporti con la pioggia Caduta. Ann. d'Igiene Speriment., IX, 3, 1899, p. 354.

August and September has no influence in the development of the primary fever, and has effect only on the recurrent form.

**The Freeing of a City from Mosquitos for the Prevention of Malaria.**—C. Ferini and S. Lumban[1] contribute a paper giving the results of experiments to rid the city of Sassari in northern Sardinia of mosquitos. They allude to several cities in ancient and modern times which had been made almost untenable by this pest. They sought out all the places in the city where the insects were bred in open wells, cisterns, drains, and pools of stagnant water. The method employed was the destruction of larvæ by the use of petroleum, applied to these collections of water twice a month. The flying insects were destroyed by means of chlorin, and in houses by mixtures of pyrethrum, chrysanthemum flowers, or the zanzoline of Celli and Casagrandi. The success of their experiments was such that Ferini concludes that it is practicable to rid of mosquitos any city where the conditions are favorable. The article is illustrated with a plan of the city of Sassari, showing the location of cisterns, pools, and collections of water. Another article by the same authors in the same journal[2] presents a list of simple and compound substances which may be employed as culicides. The former consists of animal and vegetable fats, essences, powders and vegetable infusions, waters, acids, and animal extracts. The substances entering into the compounds are mainly eucalyptus, vaselin, tar, and other articles having a pungent odor. See also other papers on malaria and mosquitos, by R. Ross,[3] and by Thin.[4] Also observations upon malaria, with experiments in the island of Asinara near Sardinia, by Ferini and Tonsini,[5] and a paper by Manson,[6] entitled "Experimental Proof of the Mosquito Malaria Theory."

**Epidemic Diarrhea.**—Newsholme[7] concludes that: (1) Epidemic diarrhea is chiefly a disease of urban life, but not necessarily having any relation to density of population; (2) as a fatal disease it is a disease of the artisan, and still more of the lower laboring classes; (3) towns having a good system of sewerage have, as a rule, much less diarrhea than those which retain other methods of sewage removal; (4) towns with the most perfect scavenging arrangements have the least epidemic diarrhea; (5) in houses having a solid rock foundation, with but little loose material, the diarrheal mortality is low; (6) given two towns equally placed, so far as social and sanitary conditions are concerned, their relative diarrheal mortality is proportional to the height of the temperature and the deficiency of rainfall in each town, especially that of the third quarter of the year. The fundamental condition favoring epidemic diarrhea is an unclean soil, the particulate poison of which infects the air, and is swallowed with food, especially milk. In other words, epidemic diarrhea, like typhoid fever, is a "filth disease." Milk is not an actual cause, but is simply a vehicle of infection.

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[1] Ann. d'Igiene Speriment., Rome, 1900, vol. x, 1, p. 93.

[2] Ann. d'Igiene Speriment., Rome, 1900, vol. x, 1, p. 89.

[3] Rev. Scientifique, June 23, 1900, 769.

[4] Brit. Med. Jour., Feb. 10, 1900, 307.

[5] Ann. d'Igiene Speriment., x, 2, 1900.

[6] Lancet, Sept. 29, 1900.

[7] Public Health, Dec., 1899, p. 139.

**Oysters and the Spread of Typhoid Fever.**—Coincident with the researches of Bulstrode in England and of Mosny in France, Uffreduzzi and Zenoni [1] reported the results of experiments to determine the viability of the typhoid germ in sea-water and in shell-fish. The chief centers of oyster production in Italy are at Taranto, Venice, Spezia, and Fusaro. (1) Experiment: Sea-water sterilized and maintained at a temperature of 15° C., typhoid bacillus added and found living at the end of a fortnight. With water from fresh oysters the bacillus was found active after 3 or 4 days. (2) Tubes of broth were inoculated with liquid from oysters sold in Milan and brought from the sea-coast. Bacillus typhosus not found, but Bacillus coli was found and was virulent. (3) Live oysters were planted in sea-water infected with Bacillus typhosus after preliminary sterilization. Bacillus found alive in water taken from mantle cavities of these oysters up to ninth day, but never in the body. Oysters may affect harmfully those that eat them in various ways: (*a*) By giving rise to true typhoid fever when they contain the specific bacillus. (*b*) By giving rise to acute gastro-enteritis by means of some poison which probably forms in the oyster after death. (*c*) There is a third form of illness produced by oysters, from a special ptomain, which is characterized by a continued fever with profound depression, lasting 1 or 2 weeks, and often ending fatally. (4) Experiments were made upon oysters undergoing simple decomposition. The filtrate was injected into the peritoneal cavity of rabbits. That of putrid oysters proved fatal in every instance; that of fresh oysters was less fatal. See also a full discussion of the same subject by Mosny.[2]

**Tuberculosis.**—Roger and Garnier [3] present the case of a tuberculous woman who died 17 days after delivery. The mammary gland was healthy. Four cubic centimeters of the milk of this woman were injected under the skin of a guinea-pig, which died on the thirty-third day with general tuberculosis. The infant nursed its mother for 2 days only, and died 6 weeks after birth. It presented tuberculous granulations in the mesentery, the liver, the spleen, and the kidneys. In this case the digestive tract appears to have been the chief, if not the only, avenue for the entrance of infection.

**The Prevention of Tuberculosis.**—Thompson, of New South Wales,[4] proposes the following measures: (1) Phthisis should be made notifiable and the register should be declared confidential as regards names; (2) the State should provide for the free examination of sputa, and this should also be confidential between medical men and the central health authority; (3) it should make the disinfection of rooms from which consumptives have been removed or in which they have died obligatory on owners and occupiers of houses; (4) district registrars should be required to notify every death from phthisis as soon as registered by them; (5) good building laws should be enacted and enforced.

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[1] Giornale della Reale Società Italiana d'Igiene, 1899. Report of Congress of Hygiene at Como, Sept. 29, 1899.

[2] Rev. d'Hyg., Dec., 1899; Jan., Feb., and Mar., 1900.

[3] La Semaine méd., Feb. 28, 1900, p. 77.

[4] Public Health, Jan., 1900, p. 248.

The central authority should forward by post some brief instructions to the persons whose names are notified. The information supplied by a well-ordered scheme of notification would in a few years furnish the requisite guide to effective action, and, by being communicated to the public, would gradually render effective action more completely possible. See also a paper by Scurfield,[1] "On the Use of Tuberculin for Lessening the Prevalence of Tuberculosis among Cattle and Children. Is This a Matter for Public Interference?" making certain suggestions for legislation upon the subject of tuberculosis among cattle, and the sale of their meat and milk as food.

**Transmission of Tuberculosis by Means of Postage-stamps.**—Busquet,[2] of Algiers, found in his hospital service a young soldier with advanced phthisis, an enthusiastic stamp collector, who was in the habit of moistening the stamps with his tongue. Busquet bought 300 stamps of him and immersed them in a pint of sterilized water for 24 hours; 8 guinea-pigs inoculated with this water became tuberculous. The author experimented upon various disinfectant solutions for the purpose of finding one which could be used for washing the stamps without injuring their colors. He gave preference to a 5% solution of carbolic acid.

**Infection by Means of Drops or Spray of Saliva.**—Koniger[3] presents the results of experiments in similar line with those of Flügge intended to show the distance to which germs of tuberculosis may be expelled from the mouth in minute drops of spray by the acts of speaking, coughing, and sneezing. In an apartment where there was no perceptible air-current germs of well-defined species were found to be carried to a distance of 7 meters (nearly 8 yards) from the individual projecting them, and to a height of 2 meters. Other valuable observations are also presented.

**Tuberculous Infection in Hospitals.**—M. Letulle[4] shows how the hospitals of Paris had proved to be the means of spreading infection among the sick, the students, and other attendants. He obtained from the Augustine fraternity of the Hotel Dieu the following figures, dating from 1876 to 1899: Out of a population of from 110 to 115, which had been maintained at about the same number from year to year, there had been in the 24 years 102 deaths, of which 82 were caused by pulmonary tuberculosis. There were 9 deaths from this cause in this brotherhood in 1890 and 7 in 1893. He notes as a probable cause that it was customary for the inmates to go through the galleries armed with brooms and feather dusters, sweeping and dusting the floors, which were abundantly soiled with sputum. This "massacre" did not cease until the authorities forbade the practice of dry sweeping and dusting.

**Tuberculous Infection of Clerks in a Library.**—S. A. Knopf[5] reports 20 clerks in the office of a board of health infected by means of saliva from a sick employee, who was in the habit of moistening the fingers in turning the leaves of books. He adds: "It is probable that in coughing, sneezing, and reading in a loud voice, he threw upon the pages of the books saliva infected with tuberculosis, a mode to which Flügge has called attention."

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[1] Public Health, Oct., 1899, p. 39.

[2] Bull. Med., 16, Dec., 1899, p. 1126.

[3] Zeit. f. Hyg. u. Inf., 34, p. 119.

[4] Presse méd., Mar. 21, 1900, p. 107.

[5] Presse méd., Feb. 24, 1900, p. 70.

**Diphtheria Bacilli in the Throats of Healthy Persons.**—Kober[1] examined cultures taken from 2 series of apparently healthy persons. The first series comprised 123 individuals known to have been recently in contact with persons suffering with diphtheria. Diphtheria bacilli were found in the throat in 10 of these (8 %). The second series comprised 600 individuals who had not recently come into contact with any cases of diphtheria, and from 5 of these a diphtheria-like bacillus was isolated. The bacillus isolated from the first series was of typical virulence for the guinea-pig; that which was isolated from the second series was not pathogenic for the guinea-pig.

**Report of Committee on Infectious Diseases in Public Schools.**[2]—Following a last exposure, provided no illness has resulted, one may return to school after a quarantine lasting as follows: In yellow fever, 5 days; influenza (grip), 5; diphtheria, 12; measles, 16; smallpox, 18; typhoid fever, 18; typhus fever, 18; chicken-pox, 19; scarlet fever, 20; German measles, 21; whooping-cough, 21; mumps, 25. When a pupil has had scarlet fever, he may return to school 7 weeks from the appearance of the eruption, if desquamation has ceased, the nose and throat are quite healthy, and all complications are over, and if disinfection of house, patient, and belongings has been thoroughly done. After measles, in 3 weeks from the appearance of the rash, if desquamation has ceased, and there is no cough, discharge from the ears, or inflammation of the eyes, and the house, clothes, and belongings of the patient have been disinfected. After mumps, at the end of 20 days, if all glandular swelling has disappeared and there is no tenderness of the breasts or other parts of the body. After whooping-cough, when all spasmodic cough or whooping has ceased, or whenever cough has entirely left. After German measles, in 2 weeks after the commencement of the disease. After smallpox, when all the scabs are off, especially of the hands and feet, everything that has come in contact with the patient has been burned, and there has been thorough disinfection of everything else. After chicken-pox, when all the scabs are off, special care being taken to see that there are no scabs on the scalp, for which purpose the hair should be kept very short and the head thoroughly washed frequently. After diphtheria, when two consecutive negative cultures from the nose and from the throat have been obtained. When no cultures are taken, a period of from 18 to 20 days should elapse after the mucous membrane of the throat has assumed a normal appearance, provided there is no discharge from the nose, before the patient is released from isolation. After yellow fever, influenza, typhoid fever, and typhus fever, at the discretion of the attending physician, who must certify in writing that all danger of contagion is over and proper disinfection of everything has been done.

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[1] Rev. des Mal. de l'Enf., July, 1900.

[2] Proc. of Mass. Med. Soc., 1899, p. 19.

**Hydrophobia in Germany.**[1]—On account of muzzling of dogs hydrophobia has rarely occurred in Germany. The deaths for 1894–98 were only 4 or 5 annually. An establishment was organized at Berlin, and from June to December, 1898, 137 patients were treated and 107 suspected animals tested for rabies. Of these latter, 95 gave positive results, and 89 of the patients had been bitten by them. From January to April, 1899, 107 patients had been treated and there were no deaths.

**Disinfection.**—An excellent paper by W. L. Mackenzie on methods of disinfection appears in the April and May numbers of "Public Health" (London, 1900). See also Sternberg's pamphlet, recently revised and published by the American Public Health Association.

**Vaccination in England.**[2]—Out of 914,205 births in England in 1896, 602,922, or 66%, were successfully vaccinated; 209,007, or 22.9%, being not finally accounted for. A total of 110,728 consecutive primary vaccinations was performed by the Board's medical officers, "without the occurrence of a single case of so-called insusceptibility."

**Soap as a Disinfectant.**—Tonzig[3] reviews the experimental work hitherto done as to the disinfecting power of soap. He says that many kinds of soap have been introduced in which a high disinfecting power is claimed on account of the addition of special disinfectants, like mercuric chlorid, salicylic acid, etc. These, he says, often destroy both the disinfectant action of the soap and of the added antiseptics themselves, as has been shown by Nijland, Pellizari, Reithoffer, and others. The author details the results of experiment with different kinds of soap and creolin upon cultures of certain disease germs. It was shown that this addition to the soap did not increase its disinfectant power, and that simple soaps of good quality were quite as valuable as, if not better than, those which were sold as special disinfectant soaps.

**Methods of Purifying Drinking-water by Chemical Substances.**—Schumburg[4] presents the results of experiments showing the effect of treating one liter of the water of the Spree with 13 different substances: namely, tea and coffee, spirituous liquors, formalin, vinegar, permanganate of potassium and lime, alum, sesquichlorid of iron, lime, oxygenated water, iodin, chlorin, and bromin. Of these, he gives preference to bromin, and in another paper, by Pfuhl,[5] the method of employing bromin for this purpose is detailed. He adds that the mixture gives to the drinking-water the taste of Seltzer water which has lost its freshness, and has no injurious effect on health.

## WATER-SUPPLY AND SEWERAGE.

**The Treatment of the Sewage of Towns.**—At the annual meeting of the German Society of Public Hygiene,[6] held at Cologne in

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[1] Hyg. Rundschau, Nov. 7, 1899.

[2] Local Gov. Rep., Suppl. of Med. Officer, 1898–1899.

[3] Gaz. d'osped. e delle clin., No. 6, 1900.

[4] Veröffentl. a. d. Geb. d. Militär-Sanitätswesen, XV, 1900.

[5] Zeit. f. Hyg. u. Inf., 1900, XXXIII, 53.

[6] Viertelj. f. öff. Gesundheitspflege, vol. XXXI, p. 136.

September, 1899, Dunbar introduced the subject of sewage treatment, with special reference to modern methods, in a series of 7 propositions, affirming that the first object was to separate the suspended and floating impurities, to eliminate the dissolved organic matters liable to undergo putrefaction, and to destroy pathogenic germs; that the requirements of hygiene, with respect to the degree of purity needed, vary in accordance with local conditions; that by means of careful irrigation it is possible to attain a high degree of purification without nuisance, and in certain cases without loss; that intermittent filtration, accompanied at times by a chemical or mechanical treatment, will insure upon a relatively small area of land a considerable degree of efficacy; that the so-called biologic system of treatment depends mainly upon intermittent filtration and the self-purifying properties of the filter when not in action; that neither mechanical disposition nor the various combined chemical and mechanical methods of sewage treatment are capable of effecting a notable decrease in the amount of dissolved organic matter contained in the sewage water; finally, that the disinfection of town sewage can be carried out more efficiently by the use of calcium chlorid than by means of any other known chemical, notably quicklime, and that clarified sewage can be much more readily and safely disinfected than raw sewage water. The author maintained these theses, and in the course of his arguments asserted that in sewage irrigation two chief aims were present—the one to render the sewage water innocuous, and the other to obtain from it those substances yielding food for vegetation.

**Disinfection of Wells by Means of Potassium Permanganate.**—Delorme,[1] surgeon in charge of a camp at Chalons, presents the results of an experiment in the disinfection of wells which had not been used for 3 years, and the water was consequently foul. Steam was first tried, without success, in consequence of inability to raise the temperature of the well-water above 30° C. By means of potassium permanganate, however, the bacteria were quickly reduced from 112,000 per cubic centimeter to 150, and the taste, which had been very offensive, was entirely changed for the better. The wells were then pumped dry, and the quantity of permanganate in the fresh water flowing in was inappreciable.

**Bacteriologic Examination of Chicago Sewage.**—Gehrmann,[2] director of the Municipal Laboratory of Chicago, reviews the results of Klein's examinations of London sewage, including his discovery of Bacillus enteritidis sporogenes, and sought for it in the sewage of Chicago. He examined 42 specimens of sewage; also 130 samples of the city water-supply and some samples of milk to which river-water had been added. From these examinations he concludes that "bacteria of the species described by Klein, or very similar to them, are practically always present in the sewage of Chicago." He states, further, that "it would not appear that search for Klein's bacillus is of great value in determining sewage pollution—at least, not from the Chicago water-supply."

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[1] Bull. Acad. de Méd., Jan. 19, 1900.

[2] Monthly Bull. Chicago Health Dept., July, 1899.

The results have practically all been negative. This has been true even during a recent period of pollution, when Bacillus coli communis was abundantly demonstrated.

**Effect of Sewage Pollution upon the River Limmat, Switzerland.**—Ascher [1] presents the results of a series of observations made at Arc and at Wettingen, two points on the River Limmat, below Zurich. Samples of water were taken simultaneously at these two points. As a result of these observations the author concludes, with Schlatter, that the so-called bacterial self-purifying process was due mainly to the action of sedimentation.

**Provision for Storm-water in Sewage Purification Works.**—Engineer Martin [2] reaches the following conclusions: (1) That no definite rule as to the proportion of storm-water to be dealt with can be applicable in all cases; (2) that the first scourings of the sewers and surfaces around should pass into the ordinary works before any overflow is allowed to take place; (3) that the works provided for dealing with storm-water should always be ready to receive it at any hour of the day or night; (4) that reasonable provision may be made for dealing with storm-water without adding unduly to the cost of the works.

**Treatment of Crude Sewage of London at the Outfall.**—The following recent statement of Clowes [3] presents the results obtained at the London outfall more definitely than they were given in the YEAR-BOOK for 1900 at p. 549: (1) The sewage was allowed to flow into large tanks which contained fragments of coke about the size of walnuts. As soon as the level of the liquid had reached the upper surface of the coke-bed, its further inflow was stopped, and it was allowed to remain in contact with the coke surface for about 3 hours. It was then allowed to flow slowly away from the bottom of the coke-bed. This outflowing liquid constituted the "sewage effluent." After an interval of about 7 hours the processes of filling and emptying the coke-bed were repeated with a fresh portion of sewage. The coke-bed was usually filled in this way twice in every 24 hours. (2) A purifying action was produced by the coke-bed. This depends upon the introduction of bacteria from the sewage, and the maintenance of the purifying action is secured by the continuous presence of bacteria or their enzymes upon the coke surfaces, and by the frequently renewed contact of these surfaces with oxygen. (3) The aeration of even the lowest portions of a deep coke-bed seems to be satisfactory in the foregoing method of working, since the air present in the interstices of the coke, between two fillings with sewage, usually contains 75 % of the amount of oxygen present in the air. (4) Raw sewage, which had been deprived of its larger particles by screening it through coarse gratings, lost practically the whole of its suspended matter by remaining in such a coke bacteria bed for 2 or 3 hours. It appears that the suspended particles of fecal matter undergo liquefaction by the bacteria, and do not collect upon the surface of the coke. (5) The sand and grit and finer mud, arising mainly from the wear of road surfaces, however, were deposited upon the coke surfaces, and gradually reduced the capacity of the coke-bed. (6) Hair, fibrous matter, woody fiber derived from the wear of wooden street pavements, and particles of chaff and straw, mainly derived from the dejecta of horses employed in the street traffic, are also deposited upon the coke surfaces and gradually choke the coke-bed. These substances, which consist mainly of cellulose, are apparently acted upon by bacteria only with extreme slowness under the foregoing conditions. They arrive, however, in a water-logged condition, and rapidly settle down from the sewage if its rate of flow is reduced. (7) In dealing with the sewage of the metropolis it seems best to allow the roughly screened raw sewage to undergo a somewhat rapid process of sedimentation, in order to allow these matters (5, 6) to subside; and then to pass the sewage direct into the coke-beds. The dissolved matters and the small amount of suspended matters which are still present in the sewage are then readily dealt with by the bacteria of the coke-bed, and practically no choking of the bed occurs. (8) The sewage effluent from the coke-bed is entirely free from offensive odor, and remains inoffensive and odorless even after it has been kept for a month. It is clear, except during heavy rain, when a turbidity is produced by fine mud particles. Many pond and river fish have been kept in this constantly renewed effluent for a month, and were found to be perfectly healthy at the end of that period. (9) The chemical character of this effluent may be briefly indicated by stating that, on an average, 51.3% of the dissolved matter of the original sewage, which is oxidizable by permanganate, has been removed by the bacteria, and that the portion which has been removed is evidently the matter which would become rapidly offensive and would quickly lead to de-aeration of the river-water if it were allowed to pass into the river. The foregoing percentage removal (51.3) was effected by coke-beds varying from 4 to 6 feet in depth. A similar bed 13 feet in depth has proved more efficient, and has for some time produced a percentage purification of 64, while an old bed 6 feet in depth has given a percentage purification of 86. A repetition of the treatment of the effluent in a second similar coke-bed has produced an additional purification of 19.3%, giving a total purification of 70.6%. It should be noticed that the foregoing purification is reckoned on the dissolved impurity of the sewage; the suspended solid matter is not taken into account. (10) The bacteriologic condition of the effluent corresponds in the main with that of the raw sewage. The total number of bacteria undergoes some reduction in the coke-beds, but the different kinds of bacteria which were present in the sewage are still represented in the effluent. (11) The introduction of such a sewage effluent into the lower Thames appears to be unobjectionable. The river-water at this part is uniformly muddy; it is always brackish, and frequently salt to taste, owing to the presence of tidal sea-water. It is, therefore, not capable of being used for drinking purposes. The effluent will certainly cause no deposit upon the river-bed, and will ordinarily tend to render the muddy river-water clearer by mixing with it.

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[1] Zeit. f. Hyg. u. Inf. 33, 1, p. 1899.

[2] Jour. Sanit. Inst., Jan., 1900, p. 624.

[3] Jour. Sanit. Inst., July, 1900, p. 308.

No offensive smell can be emitted by the effluent as it is discharged, and the bacteria which it contains will slowly and inoffensively remove the remaining dissolved organic matter from the effluent after it has been introduced into the river. The effluent will be suitable for the maintenance in the river of healthy fish life.

**Practical Results of Experiments in Sewage Treatment.**—Garstang [1] discusses the subject of sewage treatment by means of contact-beds composed of waste cinders of $\frac{1}{8}$-inch to $\frac{3}{4}$-inch diameter, claiming a rate of 2,000,000 gallons an acre a day. The purification, however, appears to be incomplete.

**The Pail System of Excrement Removal.**—This system of removal of excreta has been in use for several years in many of the cities of England, as at Manchester, Birmingham, Nottingham, Hull, and Sunderland. Armstrong,[2] Medical Officer of Health of Newcastle, issued a circular to other medical officers asking their opinion of the system. The system was condemned by these officers with scarcely an exception. This method of disposal was also considered as conducive to the spread of typhoid fever. The chief inspector of Newcastle replied that pail closets were desired only by the owners of small or tenemented houses, never by the occupiers. Their advantages are: (1) Less cost than water-closets; (2) no water-rates to pay; (3) no drain to choke or apparatus to get out of order; (4) frost does not affect them. Armstrong believes that they constitute a serious evil.

A good description of the sewage and sewage disposal of the city of Paris as it is now conducted is presented by Steuernagel, of Cologne.[3] Dunbar [4] presents papers on the nature and employment of the biologic method in the purification of sewage. See also another paper by the same author.[5]

## INDUSTRIAL HYGIENE.

**Gannister Disease.**—Birmingham [6] describes this disease as caused by the working of gannister—a compact, hard material occurring in the clay in parts of England and used in making bricks. It is ground in mills. It is so hard as to require blasting. The constant irritation of the lungs caused by breathing the fine dust leads to the formation of fibroid tissue. The mortality among gannister miners is 42 per 1000; grinders, 180 per 1000; and among brickmakers, 22 per 1000. The author submits the following preventive measures: (1) Only adults to be employed; (2) rigid medical examination before beginning work; (3) medical examination every 3 months, and all employees having bronchitis to stop work until recovery; (4) an efficient respirator advised; (5) frequent inspection of the works; (6) automatic machinery to be used when practicable; (7) water to be used to allay the dust; (8) efficient ventilation advised.

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[1] Public Health, May, 1900, p. 612.

[2] Pail-closets in Tenement Property, by H. E. Armstrong, M.D., Nov., 1899. An Appendix to his Annual Report.

[3] Centralbl. f. allg. Gesundheitspflege, XIX, 1900, p. 404.

[4] Deut. Viertelj. f. öff. Gesundheitspflege XXXI, 1899, p. 625.

[5] Viertelj. f. ger. Med. u. öff. Sanitätswesen, Suppl., 1900.

[6] Jour. Sanit. Inst., April, 1900, p. 66.

**Cataract among Workmen Exposed to Great Heat.**—Pröbsting,[1] of Cologne, as well as Meyhöfer and Hirschberg, show the results of observations among the factories of Ehrenfeld. Lesions of the crystalline lens were found among them in the ratio of 12%. Among adults over 40 years old it was as high as 24%, the degree of injury varying from light cases to complete opacity. It was especially noted among those glass workers who draw the melted glass from the furnaces. These men are subjected to a heat of 65° C. (149° F.), the left side of the face being principally exposed, and it is chiefly the left eye which suffers. Meyhöfer believes that the excessive perspiration among glass workers plays an important part in producing this injury. Another cause he finds in the brilliant white light, which dazzles the eyes of workers in glass, and by fatiguing the retina may injure the crystalline lens by reflex action.

**Oil Merchants; their Immunity against the Plague.**—In examining the history of the plague, M. Soir[2] discovered the fact that workers in oil did not contract the plague. He therefore tried the following experiment: He took a rat from a house infested with fleas, killed it, and placed it in a cage with two other rats. The skin of one of these rats had been oiled and the other was left in its normal condition. When the body of the dead rat had become cool, all the fleas migrated to the body of the dry rat, not one being found upon the oiled rat. To this aversion of fleas to oil he thought it probable the workmen owed their immunity to the plague. Ferrin and Desgenettes had made similar observations at an earlier period, and advised anointing with olive oil as a prophylactic. See also conclusions of Galli-Valerie[3] relative to the rôle played by rats and fleas in spreading the plague.

**Vanillism among Workmen.**—Andeond[4] mentions two kinds of vanillism or poisoning by the use of vanillin food, and also that which is produced among the workmen who prepare it. Among the workmen in a factory at Geneva the author observed a papulovesicular eruption of exposed parts of the body, accompanied with a burning sensation, edema of the same parts, and sometimes with boils.

**Lead Colic among Electricians.**—Talamon[5] observed at the Hôpital Bichat several cases of lead colic among the class of workmen who charge the accumulators and are required to spread red paint with the palm of the hand upon leaden plates. At the end of 3 or 4 weeks severe symptoms of lead-poisoning supervene.

**Dangerous Trades.**—Several parliamentary reports[6] have been issued, in which the dangers of grinding, file-cutting, and many other occupations, together with those incident to pottery manufacture, are treated and measures are advised for their prevention.

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[1] Centralbl. f. öff. Gesundheitspflege, 1899, p. 425.

[2] Rev. Scientifique, Mar. 31, 1900.

[3] Centralbl. f. Bakt., Jan. 6, 1900.

[4] Rev. Méd. de la Suisse Rom., Oct., 1899, No. 10.

[5] Méd. Moderne, Feb. 7, 1900, p. 83.

[6] Fourth Interim Report, and Final Report on Certain Dangerous Trades. Report on Lead Compounds in Manufacture of Pottery, London, 1899.

## SCHOOL HYGIENE.

**Medical Supervision of Schools.**—The report [1] of a committee on medical inspection of schools, made to the Public Education Association of Philadelphia, March 12, 1900, contains the following recommendations: (1) That school-houses condemned by the Board of Health as unfit for use for school purposes be closed until put in order; (2) that a maximum of 45 pupils in average daily attendance at any one school be adopted, at least for all new divisions; (3) that, beginning October 1, 1900, the fitness of the hearing and sight of all children for school work be annually tested; (4) that the use of common drinking-cups be avoided by the introduction of a new fountain system for drinking purposes, at least in new buildings; (5) That boiled water be used wherever possible for drinking purposes in schools in which adequate filtration is not employed.

**The Teeth of School-children.**—Spokes [2] gives the following figures as the result of examination of 10,500 English and Scotch boys and girls, of an average age of 12 years: These children had 37,000 unsound teeth. There were 18,000 decayed temporary teeth, more than half of which should have been "stopped." There were 19,000 permanent teeth, 13,000 of which should have been saved and 6000 required extraction. Only 14% had teeth free from decay.

**The Eyes of School-children.**—Butler [3] states the following summary: (1) Refractive errors are extremely common among school-children, being present in as high as 50%; (2) these errors are of great importance on account of the immediate symptoms and disabilities which follow their neglect; (3) they are important factors in the production of many painful and disabling affections, common enough in childhood, but still more so in adult life; (4) this early correction is demanded not merely as a means of therapeutics, but as an important measure of preventive medicine.

**Obstruction of Light by Frosted Windows in School-rooms.**—Wolpert [4] presents a series of tables in which are given the results of observations on the obstruction of daylight in winter in consequence of the frost on window-glass.

## MUNICIPAL HYGIENE.

**Street-watering.**—Mazuschita [5] examined the dust of watered and unwatered streets of Freiburg, the objects of watering being to prevent the dust from rising into the air and thus making it unhealthful and disagreeable, and also to produce cooling by evaporation of the water. He showed that the bacteria multiplied in the moist dust, but were destroyed in the dried dust by the action of sunlight. The bacteria in the watered dust exceeded those in the unwatered dust in the ratio 1,204,948 to

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[1] Phila. Med. Jour., vol. VI, p. 369, 1900.

[2] Jour. Sanit. Inst., April, 1900, p. 72.

[3] Jour. Sanit. Inst., April, 1900, p. 95.

[4] Hyg. Rundschau, Jan. 1, 1900, vol. x, No. 1, p. 3.

[5] Arch. f. Hygiene, 35, 1899, p. 252.

589,857. In good weather and at the end of 4 days of good dry weather these numbers had become respectively 2,211,500 and 1,893,000, but at the end of 26 days they were reduced to 97,333 and 37,250; that is to say, the reduction of germs in the dust amounted to 95.6% and in the dried dust to 98.1%. The writer concludes that street-watering is not a sanitary measure—a conclusion which appears to have been hastily made, since the object secured by street-watering is the fixing of the germs upon the ground in such a manner that the air above them shall be pure and respirable.

**Disinfection and Street-watering.**—Experiments at the Thompson-Yates Laboratories[1] demonstrated the superiority of "chloros" over carbolated creasote for use in street watering-carts. Bacillus coli communis in pure culture in proportion of about 100 organisms per cubic centimeter was entirely destroyed after 15 minutes' exposure to a solution of chloros (1 : 10,000), while with carbolated creasote (1 : 2500) there was no appreciable reduction. With street mud there was a similar effect, but not quite so decided, the carbolated creasote causing no reduction in a 1 : 5000 solution, while the "chloros" in 1 : 10,000 rendered the mud nearly sterile.

**Underground Bakehouses.**—The chief objections to underground bakehouses are well summarized by E. W. Hope,[2] Medical Officer of Health, Liverpool, as follows: (1) The difficulty in obtaining proper fall for draining when cleansing the bakehouse floors; (2) risks from backing up of drains in time of flood or from choking of drains; (3) the difficulty of obtaining adequate light and ventilation, owing to the way in which the ovens are usually arranged; (4) the risk of dust and other refuse being blown into the bakehouse, and on to the tables, etc., owing to the windows being nearly on a level with the street; (5) the difficulties in preventing the areas from becoming receptacles of rubbish and filth.

**Baths and Bathers in London.**[3]—Under the Baths and Washhouses Acts, 2532 public baths are in existence—887 first class, 1659 second class, and 85 swimming baths. The total number of bathers in 1897–98 was 4,463,109. The finances showed a deficit of £83,360 ($333,000).

## ISOLATION HOSPITALS.

**Difficulties of Administration.**—Goodall[4] discusses in full the difficulties of administration. Those which relate to the public are becoming fewer each year. The proportions of admissions to notifications has notably increased. One difference between this class of hospitals and general hospitals consists in the fact that the patient who enters a general hospital goes voluntarily, as a rule, but removal to an infectious disease hospital usually takes place only after much persuasion; hence prejudice against these often arises. In a general hospital the patient can usually leave when he chooses to do so, but in the isolation hospital the patient should be allowed to leave only when the public health and safety would permit. Visiting such hospitals should be forbidden. The difficulties that arise with the general practitioner are usually due to errors in diagnosis. Great discretion is necessary on the part of the admitting physician in regard to doubtful cases. The author also discusses the difficulties which arise from the nature of the hospital itself.

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[1] Public Health, Aug., 1900, p. 810.

[2] Sanit. Rec., Aug. 31, 1900.

[3] Returns of Local Government Committee to the London Council, Nov. 21, 1899.

[4] Public Health, June, 1900, p. 648.

**New Form of Isolation Ward.**—Roux [1] has introduced in the children's hospital at Paris a series of separate rooms.

## TENEMENT HOUSE HYGIENE.

**Housing of the Working Classes.**—At the meeting of the Sanitary Institute Congress [2] at Southampton, in August, 1899, it was stated there were in Liverpool 70,000 people living in unsanitary houses. The city authorities could not turn all these people out all at once, since there was no way of rehousing them. The existing laws providing for such cases were expensive, and they obtained a special act. Under this act they dealt with about 500 houses a year, and had already demolished 4200, while 800 had been erected, either by the corporation or by private enterprise. The new houses were not taken by the people who were displaced, who were, therefore, bettered but little, if at all. They then set about erecting a series of houses which could be let at one shilling or one shilling and sixpence a room. They had erected 180 such tenements, and the poorest of the persons displaced had the first offer. The houses were not all that one would like, but were a great improvement on what the people had previously occupied.

## DISPOSAL OF THE DEAD.

**Growth of Sentiment in Favor of Cremation in America.**—Louis Lange,[3] President of the Cremation Society of New York city, furnishes the following figures: The number of crematories in existence in the United States, March 1, 1900, was 26. The number of bodies incinerated in these establishments in each year since the building of the first crematory by Lemoyne, at Washington, Pa., is as follows:

| YEAR. | BODIES. | YEAR. | BODIES. | YEAR. | BODIES. |
|---|---|---|---|---|---|
| 1876 to 1883 | 25. | 1889 | 249. | 1895 | 1017. |
| 1884 | 16. | 1890 | 379. | 1896 | 1101. |
| 1885 | 47. | 1891 | 471. | 1897 | 1391. |
| 1886 | 114. | 1892 | 561. | 1898 | 1699. |
| 1887 | 127. | 1893 | 674. | | |
| 1888 | 190. | 1894 | 831. | | |

Total, 8885.

More recent information brings the total up to about 10,700 at the close of 1899.

**Cremation in Scotland.**[4]—In order to render cremation more popular, the directors of the Scottish Society have decided to issue certificates, for 5 or 6 guineas each, entitling the holder to arrange for a cremation at any time, and they hope that by this means the time may soon come when such certificates may be made available at any other crematorium, not only in Great Britain, but also abroad. By such a policy the ashes of those dying at a distance from home—as, for example, in the colonies—might be brought home and laid to rest in the family burying-place.

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[1] Presse méd., Feb. 21, 1900.

[2] Public Health, Oct., 1899, p. 29.

[3] Hyg. and State Med. in the United States, 1900, p. 59.

[4] Sanit. Rec., Feb. 16, 1900.

**Premature Burials.**—Weidman [1] takes the ground that living interments are of very rare occurrence. He quotes Dr. Prime, editor of the New York "Observer," a religious newspaper, who made careful inquiry of citizens in places where premature burials were said to have occurred, and "declared that he had never been able to verify a single case, even after having interrogated relatives and others reported to have witnessed such scenes." Weidman also adds: "Inquiry among the funeral directors and superintendents of cemeteries in my neighborhood has failed to elicit a suspicion of such occurrence, nor has there been anything noticed after exhumation that differed inside the coffin from that seen at the time of interment."

**The Relation of Bacteria to the Burial of Human Bodies.**—Klein's [2] observations assure us that but little danger may be apprehended from the burial in the soil of persons who have died of infectious diseases. The dissolution of such bodies appears to be effected by means of microbes harmless to man.

## FOOD AND DRUG INSPECTION.

The State of Indiana has enacted a law relative to the inspection of food [3] and conferred unusual power upon its Board of Health by the following provision: "The State Board of Health shall prepare rules and ordinances where and when necessary, regulating minimum standards for food and drugs, defining specific adulteration, and determining the proper methods of collecting and examining drugs and articles of food." Under this provision the Board has published standards for the following articles of food: milk, butter, margarin, cheese, coffee, tea, candy, cider, flour, jellies and preserves, honey, lard, molasses and syrups, olive oil, spices, vinegar, baking powders, and spirituous liquors and wines. The use of preservatives is also regulated. Milk must contain no added coloring-matter or preservative. It must not be sold if taken within 4 days after calving, or within 21 days before expected calving; nor must it be sold if taken from a cow fed with damaged food, or such as will impart a disagreeable flavor. It must not be taken from a sick or diseased cow, nor from one to which polluted water has been given, nor from one which has been "kept under conditions contrary to the rules of the State Board of Health governing dairies."

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[1] Lehigh Val. Med. Mag., Aug., 1900.

[2] Twenty-eighth Ann. Rep. Local Gov. Board, 1899.

[3] Brit. Food Jour., Aug., 1900, p. 224.

**A New Milk-preserving Process.**—A new process of preserving milk[1] is being tried, the invention of two Austrians, the milk being pressed in the form of a powder. The milk is taken direct from the cow and placed in a vessel at the temperature of 40° C. until the water is all evaporated. The residue is then further dried and is sealed in cans. In order to increase its keeping quality and to make it more soluble, a small quantity of an alkaline carbonate is added to it before it is evaporated, which, combining with the fat, forms a compound easy of solution in water.

**Bacteria in Milk and Other Foods.**—Bloch[2] emphasizes the fact that the harmfulness of food does not depend on the number of germs contained, but on their kind. Preussnitz[3] estimates that butter contains from 10 to 20 millions of germs to the gram. See also Keith,[4] who visited several dairies and compared the bacteriologic results and the conditions found at the dairies. He says: "On the whole, a bacterial analysis of a dairy reveals its character and 2 (examinations of milk) of the same dairy on different days constitute an almost perfect indicator of what to expect of its condition." He sums up his conclusions as follows: (1) Milk in its natural state is free from bacteria, and consequently will keep indefinitely; (2) fore milk contains a few bacteria and should be rejected; (3) milk is seeded with bacteria from dust, contact with improperly cleaned utensils, water, etc.; (4) bacterial analysis of milk may be depended upon to indicate the care it has previously had; (5) a large part of the milk examined shows it to have had proper care; (6) cooling milk does not entirely prevent growth of bacteria, but prevents souring; (7) the cans of the local dealers into which milk is put add a considerable number of bacteria to the milk; (8) a bacterial examination of dairy-milk is a valuable assistance in improving the condition of a milk-supply.

**The Standard of Pure Milk.**—There is no legal standard of milk in England.[5] The Society of Public Analysts recommends 8.5% of nonfatty solids and 3% of fat. The Somerset House Laboratory employs the same. The analysts of 13 metropolitan districts also employ this same standard, while 13 other districts employ a lower standard—namely, 8.5% of nonfatty solids and 2.75% of fat. The standard of milk in Massachusetts is variable (Acts of 1899, Chap. 223), since it requires 9.3% of nonfatty solids in the winter months (October to March inclusive) and only 9% in the remaining months, the requirement for the fat being also 3.7% and 3% respectively. The State Board of Health of Indiana has recently adopted a fixed standard of 12% for the total solids of milk, of which 3% must be fat.

**The Healthfulness of Milk Produced upon Sewage Farms.**—Andrews[6] says: "The production of milk and butter is one of the most profitable or least unprofitable of the uses to which a sewage farm can be put. The large crops of Italian rye grass and of marigold and other roots which can be raised are, as a rule, far more advantageously used on the spot for the feeding of cattle than sold to outside buyers. Hence most large towns which treat their sewage on the land devote much attention to dairy produce." He cites Birmingham, Reading, and Croydon as examples, and says, in conclusion: "From these considerations it may be asserted that, although dairy produce from sewage farms has been consumed for many years in this country on so extensive a scale that baneful effects resulting from its consumption can hardly have failed to have become manifest, such effects are in no way demonstrated. Neither in any individual case, nor from statistical evidence, has it been shown that the conditions existent on the Aldershot Sewage Farms, and especially the conditions under which the dairy operations are carried out, are materially worse than those obtaining on other well-managed farms. There would appear to be no *a priori* reason for supposing the dairy produce dangerous for human consumption."

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[1] Food and Sanitation.

[2] Berl. klin. Woch., Jan. 22, 1900.

[3] Grundzüge der Hygiene.

[4] Jour. of Mass. Assoc. Boards of Health, April, 1900, p. 23.

[5] Brit. Food Jour., Dec., 1899, p. 355.

[6] Report on the Condition of the Aldershot Camp Sewage Farm, and of the Dairy Maintained upon It, made to the Secretary of State for War, May 5, 1899.

**Diseases of Cows Communicable through Milk to Human Beings.**—Sessions[1] (veterinary) enumerates the following diseases as having been charged with being transmitted from the cow through her milk: Rinderpest, foot and mouth disease, anthrax, tuberculosis, scarlet fever, diphtheria, typhoid, rabies, tetanus, cowpox, smallpox, actinomycosis, ringworm, and diarrhea. He rejects all the foregoing, except tuberculosis and diarrhea, as being of no practical importance, and does not consider the noted Herndon case of bovine scarlet fever as established. Typhoid fever is not a disease of cows, and anthrax is not liable to be transmitted through the milk, since it does not appear in the milk until just before or after the death of the animal. See also experiments of Agricultural Station of Connecticut[2] upon the feeding of calves with the milk of tuberculous cows without infecting the former. Also bacteriologic examination of the milk of tuberculous animals.[3]

## POISONS.

**The Sale of Poisons.**—The Lords of the Privy Council[4] have approved a resolution passed by the Council of the Pharmaceutical Society of Great Britain declaring that liquid preparations of **carbolic acid** and its homologs containing more than 3% of those substances (except any preparation intended for use as sheep-wash, or any other purpose in connection with agriculture or horticulture, and contained in a close vessel, distinctly labeled with the word "Poisonous," the name and address of the seller, and a notice of the agricultural or horticultural purpose for which the preparation has been made) should be deemed poisons within the meaning of the Pharmacy Act of 1868, and should be deemed poisons in the second part of Schedule A of the said Pharmacy Act, 1868.

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[1] Jour. Sanit. Inst., April, 1900, p. 140.

[2] Ann. Rep. of Storrs. Agricultural Station.

[3] Jour. Brit. Farmers' Dairy Assoc., quoted in Brit. Food Jour., Nov., 1899, p. 331.

[4] London Gaz., Aug. 1, 1900.

Poisoning of aeronauts by **arseniureted hydrogen** was noted by Maljean,[1] who observed several cases of an obscure nature among a company of aeronauts. The hydrogen used for inflating the balloons was made from zinc and sulphuric acid. The acid was obtained from pyrites containing from 2% to 5% of arsenic.

**The Action of Mold upon Arsenic and Its Compounds.**—Abel and Buttenberg[2] have confirmed the experiments of Gosio and others. They employed the same species of mold, but made their cultures upon dough instead of potato, at a temperature of 37° C. The reaction is so delicate as to give the characteristic odor of arsenic in the presence of 0.00001 of a gram and even 0.000001 of a gram of arsenic. Experiments were made upon skins and furs, colored papers, paints, colored crayons, preserved meats, and dead bodies, and upon hair and urine of persons exposed to arsenical poisoning. Gosio's method is very simple and admits of rapid and accurate experimentation.

## RAILWAY HYGIENE.

**Formaldehyd as a Disinfectant for Railway Cars.**—Owens,[3] Chief Engineer Ill. Central R. R. Co., reports a series of experiments with formaldehyd sprayed upon sheets in a room of 500 cubic feet capacity, as a result of which he issued the following instructions for its application to passenger and freight cars: (1) Suspend the two sheets found in the outfit by their edges from the roof of the car or the bell-cord by means of the clothes-pins. The sheets should hang their full length and be placed so as equally to divide the space in the length of the car. (2) Close the doors and windows. (3) Saturate both sheets with spray from the bottle, standing with the nozle about eight feet from the sheet and throwing the spray against it, with the left hand directing the nozle, supporting the tube where it passes from the bottle, and working the bulb vigorously and rapidly with the other hand. Begin at the top of the sheet, and use half of the required quantity of fluid on each sheet. (4) For disinfecting passenger and baggage cars, fill the spray bottle with the formaldehyd solution furnished. For disinfecting freight cars, use the bottle half-full. (5) As formaldehyd is quite irritating to the hands and eyes, those using the solution are instructed to exhaust the spray quickly, leaving and closing the car as quickly as possible. Caution should be exercised not to get the liquid on the hands or in the eyes, and not to inhale the vapor any more than is absolutely necessary. (6) Leave the cars locked tightly for at least 5 hours after spraying and, if possible, allow 8 hours to elapse before opening. (7) On opening, take down the sheets and clean the car in the usual way. If any spots are left on furniture or polished woodwork by the formaldehyd, wipe them off with a wet towel and use furniture polish. If the sheets are still moist, they should be dried in the open air, but not washed until sufficiently soiled. (8) For disinfecting spaces other than cars, such as baggage rooms, etc., the same method may be used, employing one bottle of the formaldehyd for every 4000 cubic feet of room space, and one sheet for each 2000 cubic feet.

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[1] Arch. de Méd. Milit., Feb., 1900, p. 82.

[2] Zeit. f. hyg. u. Infektionskrankh., 1899, 32, p. 449.

[3] Jour. Am. Med. Assoc., Mar. 3, 1900, p. 518.

Blume[1] summarizes measures for the safety and health of travelers and employees on railways in 5 propositions, as follows: (1) The healthfulness of stations and sanitary conditions of the material in use; (2) rapidity of assistance in cases of accident or illness; (3) sanitary supervision of all employees according to their different occupations; (4) hospitals for the care and treatment of sick and injured employees; (5) creation of a medical staff, well organized and under the direction of a chief surgeon. He adds the reassuring statement that a traveler in Prussian railways at an average of 21 kilos an hour (about 13 miles) day and night runs the risk of being injured only after 307 years' travel, and of being killed only after 1540 years.

## HEALTH LEGISLATION.

**English Health Legislation of 1899.**—The acts of a sanitary character enacted by the British Parliament in 1899, according to the Sanitary Record,[2] were 13 in number, but most of these were of minor importance from a health point of view, except the following: The Metropolis Water Act (62 and 63 Vict., Chap. 7); the Sale of Food and Drug Act (62 and 63 Vict., Chap. 51); the Infectious Disease Notification Extension Act (62 and 63 Vict., Chap. 8). The Water Act is intended to prevent the recurrence of a water famine in London by requiring the small companies supplying the city with water to submit plans for supplying each other in time of need. The Sale of Food and Drug Act is intended not so much to protect the public health as to aid the agricultural interests of the country. The principal features are that the Commissioners of Customs are to take samples of consignments of imported articles of food for analysis. The Board of Agriculture is also authorized to take samples for analysis, and, in default of any local authority, to enforce the acts in such authority's district and at the authority's expense; the law relating to margarin is also increased in stringency, and the penalties for offenses under the Food and Drug Acts and margarin laws have been so much increased in severity as to render them likely to act as effective deterrents against fraud. The Infectious Disease Extension Act extends to the whole of England and Wales, after January 1, 1900, the provisions of the Act of 1889. The former act had practically been an act of local option, but the system had been adopted in districts having 28,000,000 inhabitants out of a total of 30,000,000. It has proved very helpful to sanitary authorities in dealing with outbreaks of infectious disease.

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[1] Die öffentliche Gesundheitspflege in Eisenbahnbetriebe, p. 76.

[2] Diary and Year-book for 1900, p. 13.

## THE HYGIENE OF CHURCHES.

**Holy Water as a Source of Infection.**—Abba[1] calls attention to the possibility of infection from the holy water used in the churches of Turin. He believed that the principles of hygiene should be applied to the churches, not only in the matter of holy water, but also that such unsanitary customs as the kissing of sacred objects or relics should be forbidden. Casagrandi and Mazza, at Rome and Turin, had examined the portions of statues which were exposed to the lips of people and found there the bacilli of tuberculosis, diphtheria, and other diseases. The author describes two appliances, one a Dutch and the other an Italian invention, for supplying a quantity of pure water free from contamination. A letter from the bishop of Fano is added in which certain measures are recommended as founded upon the observations of Abba. These are mainly the use of sawdust moistened with a sublimate solution for use in sweeping the floors, wiping away dust with moistened cloths, and washing the fittings of the confessional and the bowls or basins for the holy water with boiling water. His recommendations show that there should be no incompatibility between ritualism and hygiene. See also an excellent article by Remlinge[2] entitled "Les Églises au point de Med. de l'hygiene," in which the author discusses the hygiene of churches, fire protection, the transmission of infectious diseases, preventive means (the ideal church edifice), and certain peculiarities of the church buildings of different sects.

## INFANT MORTALITY.

Smith[3] quotes the coroner of the largest district of London, who says that out of 2319 **deaths from suffocation** in England and Wales, 2034 were under 1 year and 1340 were less than 3 months. He says that drunkenness is not, as commonly asserted, the general cause of this mode of death. On the contrary, it usually happens that the mother has slept heavily from being overtired.

Hope[4] states the **infantile death-rate of Liverpool** for 1899 as varying from 136 to 274 per 1000. In 1082 families where the children died of diarrhea, out of 4574 children born, 2229 had died, representing 509 deaths per 1000, or 5 times the normal standard. Out of 2000 infants who had died, 1602 had been insured in burial societies.

## VITAL STATISTICS.

**Birth-rates and Death-rates per 1000 Inhabitants of Different Countries and States.**—The following table presents the birth and death rates of the principal European countries for the 27 years 1871–1897, and those of each of the New England States for the 5 years 1893–1897, together with those of the single year 1898. The highest birth-rates for the 27-year period were those of Hungary, 43.1; Austria, 38.3; and the German Empire, 37.6. The lowest were those of Vermont, 21.0; New Hampshire, 21.1; and Maine, 21.5. The highest death-rates in the same period were those of Hungary, 35.2; Austria, 29.7; and Italy, 27.6. The lowest were those of Maine, 16.3; Vermont, 16.8; and Norway, 16.8.

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[1] Rivista d'igiene e sanita pubblica, Mar. 1, 1900, p. 153.

[2] Rev. d'Hyg., July, 1900, p. 577.

[3] Jour. Sanit. Inst., April, 1900, p. 176.

[4] Sanit. Rec., Aug. 24, 1900, p. 161.

BIRTH AND DEATH RATES OF DIFFERENT STATES AND COUNTRIES.

| STATES, ETC. | BIRTH-RATES. | | DEATH-RATES. | |
|---|---|---|---|---|
| | 27 Years, 1871-97. | 1898. | 27 Years, 1871-97. | 1898. |
| England and Wales | 33.0 | 29.4 | 19.8 | 17.6 |
| Scotland | 32.9 | 30.8 | 20.0 | 18.4 |
| Ireland | 24.4 | 23.2 | 18.1 | 18.1 |
| Denmark | 31.4 | 30.5 | 18.7 | 15.6 |
| Norway | 30.6 | 30 3 | 16.8 | 15.2 |
| Sweden | 29.1 | —— | 17.3 | —— |
| Austria | 38.3 | 36.2 | 29.7 | 24.9 |
| Hungary | 43.1 | 37.7 | 35.2 | 27.9 |
| Switzerland | 29.2 | 29.4 | 21.6 | 18.9 |
| German Empire | 37.6[1] | 36.2 | 25.1[1] | 20.6 |
| Holland | 34.6 | 31.9 | 21.6 | 17.0 |
| Belgium | 30.5 | 28.6 | 20.9 | 17.6 |
| France | 24.1 | 22.1 | 22.5 | 21.2 |
| Italy | 36.9 | 33.8 | 27.6 | 23.1 |
| | Five Years, 1893-97. | 1898. | Five Years, 1893-97. | 1898. |
| Maine | 21.5 | | 16.3 | |
| New Hampshire | 21.1 | | 18.0 | |
| Vermont | 21.0 | | 16.8 | |
| Massachusetts | 27.8 | 27.4 | 19.2 | 17.5[2] |
| Rhode Island | 25.8 | | 18.7 | |
| Connecticut | 24.0 | | 17.6 | |
| *New England* | 25.3 | | 18.3 | |

**Vital Statistics of England, 1899.**—The advance sheet recently issued from the Registrar-General's office of England presents a condensed abstract of the marriages, births, and deaths of that country for the year 1899. These figures will probably differ but very little from the full and revised figures which are usually issued during the following year. The **marriages** were 261,963, and the marriage-rate (persons married) was 16.5 per 1000 of the living population. The **births** were 928,640, and the birth-rate was 29.3 per 1000. Of these, 473,196 were males and 455,444 were females, or in the ratio of 1039 males to 1000 females. The **deaths** were 581,824, and the death-rate was 18.3 per 1000. Of these, 299,664 were males and 282,160 were females. The death-rate of males was 19.5 and that of females 17.2 per 1000, or in the ratio of 1133 males to each 1000 females in equal numbers living. These figures for the general population, as well as for the sexes, are estimated upon the basis of an equal rate of growth with that which had been maintained between the two preceding census enumerations of 1881 and 1891.

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[1] NOTE.—German Empire, 26 years.

[2] NOTE.—1899, 17.4.

**The Effect of the Exclusion of Certain Diseases as Causes of Mortality.**—Hayward [1] ("The Life-table of England and Wales as Modified by the Supposed Exclusion of Certain Causes of Mortality," by T. E. Hayward, M.D.) says that the most exact and vivid way of bringing into view the effects produced by any one disease is to construct a life-table based upon the supposition that this particular disease has been altogether eliminated. "What would really happen," he says, "if some disease, say phthisis, were entirely abolished, it is difficult to say, for in nature the destruction of one kind of pest, whether animal or vegetable, sometimes leaves the ground free for the more active development of other species. However, as a working hypothesis, we must consider that, if one cause of mortality be done away with, the remaining causes would continue to act with proportionate intensity on those saved from the action of the cause eliminated." Having worked out these life-tables on this assumption of the elimination of certain diseases, the author goes on to measure the effects of such diseases by answering these questions: (1) How much greater would be the chance of living from one year to the next at each age, supposing a given disease to be eliminated? (2) How many more survivors would remain at each age out of a given number supposed to be born? (3) How much longer would be the expectation of life at each age? The diseases considered are phthisis, the whole group of tuberculous diseases, cancer, typhoid fever, scarlet fever, diphtheria, diarrhea, measles, and whooping-cough. We have selected the following figures from the columns entitled "Expectation of Life" to show the difference which would be produced by excluding or eliminating entirely each of certain diseases. The accompanying table may be read as follows, for example: If cancer was eliminated entirely from the causes of death in England, the expectation of life of a boy at birth would be lengthened 0.39 of a year, or 142 days, and that of a girl at birth 0.83 of a year, or 303 days. The expectation of a young man at 25 would be increased 0.54 year, and that of a young woman of the same age, 1.14 years.

DIFFERENCE PRODUCED UPON THE EXPECTATION OF LIFE AT BIRTH AND AT 25 YEARS OF AGE BY EXCLUDING EACH OF THE FOLLOWING DISEASES:

| | Phthisis. | All Tubercular Diseases. | Cancer. | Typhoid Fever. | Scarlet Fever. | Diphtheria. |
|---|---|---|---|---|---|---|
| Males at birth . . . . | +2.58 | +3.86 | +0.39 | +0 30 | +0.53 | +0.24 |
| Females at birth . . . | +2.57 | +3.72 | +0.83 | +0.29 | +0.57 | +0.28 |
| Males at 25 years . . | +2.58 | +2.73 | +0.54 | +0.17 | 0.00 | 0.00 |
| Females at 25 years . | +2.19 | +2.29 | +1.14 | +0.15 | 0.01 | 0.00 |

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[1] Public Health, Dec., 1899, p. 214.

A similar result is shown in the table of survivors in Massachusetts, comparing the life-tables of 1855 and 1895, 40 years apart, in consequence of the lessening in the mortality from infectious diseases in the early years of life. In this table it appears that out of 10,000 infants at birth, 806 more were living at the age of 25, according to the table of 1895, than were alive at the same age according to the table of 1855.[1]

**The Fatality of Certain Diseases: Of a Certain Number Attacked, What Percentage Dies?**—The following extract[2] comprises the substance of the returns to certain inquiries sent out by the United States Commissioners to the Paris Commission of 1900, and compiled under their direction: In consequence of inquiries sent out in 1899, information was secured in regard to 619,765 reported cases of smallpox, typhoid fever, scarlet fever, diphtheria, and measles which occurred in the years 1894, 1895, 1896, 1897, and 1898, together with 75,715 registered deaths from these diseases which occurred in the same years. These were reported by the following States and cities:

STATES.

| | | | |
|---|---|---|---|
| Massachusetts | 1894–98 | Vermont | 1896–97 |
| Michigan | 1894–98 | Connecticut | 1898 |
| Rhode Island | 1894–98 | Indiana | 1898 |

CITIES.

| | | | |
|---|---|---|---|
| New York | 1894–98 | Reading | 1894–98 |
| Chicago | 1894–98 | Hudson Co., N. J. | 1894–98 |
| Philadelphia | 1894–98 | Cincinnati | 1894–97 |
| Pittsburg | 1894–98 | St. Louis | 1894–97 |
| Cleveland | 1894–98 | Baltimore | 1894–97 |
| New Orleans | 1894–98 | Milwaukee | 1894–97 |
| Minneapolis | 1894–98 | Rochester | 1894–97 |
| St. Paul | 1894–98 | Denver | 1894–96 |
| Buffalo | 1894–98 | San Francisco | 1898 |
| Toledo | 1894–98 | | |

The results of the returns received from the foregoing States and cities are as follows:

| Diseases. | Reported Cases. | Registered Deaths. | Fatality (Percentage). |
|---|---|---|---|
| Smallpox | 9,222 | 2,385 | 25.8 |
| Typhoid fever | 69,758 | 13,284 | 19.0 |
| Diphtheria and croup | 195,783 | 44,411 | 22.7 |
| Scarlet fever | 117,847 | 9,211 | 7.2 |
| Measles | 217,755 | 6,424 | 2.8 |
| Total | 619,765 | 75,715 | |

These results agree fairly well with those of the English Local Government Board for the 8 years 1890–1897, which showed a fatality for typhoid fever of 18.05%, for diphtheria of 23%, and for scarlet fever of 4.9%. The use of antitoxin for the treatment of diphtheria became general in the early months of 1895, throughout the country. If, therefore, the returns for the year 1894 are treated separately, it appears that there were 25,844 reported cases and 7654 deaths in that year, the fatality being 29.6 %, while the fatality of the remaining years was only 21.6 %. Treating the year 1898 in the same manner, the fatality was only 20.5 %, or the ratio of 31,494 cases to 6471 deaths. In 2 States and 7 cities combined, having a total population of 4,250,000, the fatality from diphtheria in 1894 was 29.7 %, and in the same places in 1898 it was only 14.6 %, confirming the statement that the diphtheria fatality has been cut in twain since the general introduction of the antitoxin treatment. It is also quite noteworthy that in several large cities, situated a thousand miles apart, the diphtheria fatality before 1895 was quite uniformly from 29 % to 30 %.

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[1] Rep. State Board of Health of Mass., 1898, p. 818.

[2] Public Hygiene and State Medicine in the United States, 1900, p. 21.

**Smallpox in European Countries.**[1]—The following figures represent the smallpox mortality in the principal European countries in 1898:

| | Deaths from Smallpox. | Death-rate per 100,000 Inhabitants. |
|---|---|---|
| German Empire | 15 | 0.03 |
| Austria | 138 | 3.64 |
| Switzerland | 1 | 0.76 |
| Principal Belgian towns | 61 | 2.57 |
| France | 57 | 0.66 |
| England | 13 | 0.12 |
| Holland | 2 | 0.14 |

In the same year the deaths from smallpox in the large cities of Europe were as follows: London, 1; Paris, 5; Berlin, 0; Hamburg, 0; Munich, 1; Cologne, 0; Liverpool, 2; Leeds, 2; Madrid, 26; Moscow, 145; Odessa, 42; St. Petersburg, 89; Rome, 3; Warsaw, 313.

**Deaths from Snakes and Wild Animals in India.**[2]—The deaths from this cause in India in 1898 were 25,166, of which 21,921 were due to snakes. At least 80,000 cattle lost their lives from the same cause.

**Statistics of Hospitals of Different Classes.**—The following figures are from the Report of the Minister of Agriculture, Industry, and Commerce of Italy, entitled "Statistica dei Ricoverati in Ospedali Pubblici e privati e in altri istituti di Assistenza," Nell' Anno, 1898, Rome, 1900, p. XXI, Confronti internationali.

## HOSPITAL STATISTICS.

### FOUNDLING HOSPITALS.

| Country. | Year. | Present Jan. 1st. | Admitted. | Discharged. | | Remaining Dec. 31st. |
|---|---|---|---|---|---|---|
| | | | | Total. | By Death. | |
| Italy | 1898 | 100,418 | 21,307 | 21,504 | 10,127 | 100,221 |
| France | 1897 | 101,474 | 18,703 | 15,800 | 3,223 | 104,377 |
| Austria | 1896 | 28,532 | 11,099 | 10,425 | 4,935 | 29,206 |

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[1] Medizinal-statistische Mittheilungen Kais. Gesundheitsamte, 1900, p. 101.

[2] Brit. Med. Jour., Nov. 25, 1899, p. 1494.

## GENERAL HOSPITALS.

| Country. | Year. | Number of Hospitals. | Inmates, Jan. 1st. | Admitted. | Discharged. | | Remaining Dec. 1st. | Remaining in Hospital per Million Inhabitants. | Deaths per 100 Discharged. |
|---|---|---|---|---|---|---|---|---|---|
| | | | | | Total. | By Death. | | | |
| Italy . . . | 1898 | 1210 | 36,550 | 436,350 | 436,199 | 43,363 | 36,701 | 13,816 | 9.9 |
| France . . | 1897 | 1234 | 52,468 | 518,372 | 517,084 | 48,983 | 53,756 | 13,512 | 9.5 |
| Austria . | 1896 | 613 | 27,443 | 386,119 | 384,592 | 32,940 | 28,970 | 15,431 | 8.6 |
| Hungary . | 1898 | 327 | 16,207 | 204,905 | 205,674 | 13,956 | 15,438 | 10,904 | 6.8 |
| Prussia . | 1897 | 1730 | 55,657 | 590,962 | 590,021 | 40,745 | 56,598 | 18,077 | 6.9 |
| Bavaria . | 1898 | 460 | 7,387 | 127,376 | 127,021 | 5,333 | 7,742 | 21,900 | 4.2 |
| Sweden . | 1897 | 107 | 3,030 | 41,972 | 41,655 | 2,095 | 3,347 | 8,354 | 5.0 |
| Holland . | 1896 | 93 | . . . | 43,651 | . . . | . . . | . . . | 8,919 | . . |

## MATERNITY HOSPITALS.

| Country. | Year. | Number of Hospitals. | Number of Births. | | Proportional Ratios. | |
|---|---|---|---|---|---|---|
| | | | Admitted. | Deaths. | Admitted per 100,000 Births in Same Country. | Death per 1000 Admissions. |
| Italy . . . . . . . . | 1898 | 135 | 16,567 | 260 | 1503 | 15.7 |
| Austria . . . . . . . | 1896 | . . | 20,496 | 158 | 2161 | 7.7 |
| Prussia . . . . . . . | 1896 | 234 | 18,945 | 264 | 1598 | 13.9 |

## INSANE HOSPITALS.

| Country. | Year. | Number of Patients Treated. | | | | | |
|---|---|---|---|---|---|---|---|
| | | Present. Jan. 1st. | Admitted During the Year. | Discharged. | | Remaining Dec. 31st. | Remaining per 1,000,000 Inhabitants. |
| | | | | Total. | By Death. | | |
| Italy . . . . . | 1898 | 33,598 | 18,420 | 17,216 | 5374 | 34,802 | 1099 |
| France . . . . | 1897 | 62,709 | 24,959 | 23,029 | 7512 | 64,639 | 1689 |
| Switzerland . . | 1898 | 6,418 | 2,525 | 2,303 | 532 | 6,640 | 2128 |
| Belgium . . . | 1897 | . . . | . . . | . . . | . . | 13,568 | 2060 |
| Holland . . . | 1896 | 7,319 | 1,638 | 1,419 | 619 | 7,538 | 1540 |
| Austria . . . . | 1896 | 12,170 | 8,274 | 7,718 | 2144 | 12,726 | 511 |
| Hungary . . . | 1898 | 2,064 | 1,711 | 1,319 | 442 | 2,456 | 130 |
| Prussia . . . . | 1894 | 39,308 | 19,403 | 16,689 | 3975 | 42,022 | 1346 |
| Bavaria . . . . | 1898 | 5,258 | 1,946 | 1,686 | 439 | 5,518 | 951 |
| Sweden . . . . | 1897 | 4,140 | 880 | 779 | ? | 4,241 | 851 |
| Massachusetts . | 1899 | 6,977 | 2,954 | 2,529 | 726 | 7,402 | 2800 |

## BLIND AND DEAF-MUTE ESTABLISHMENTS.

| COUNTRY. | YEAR. | NUMBER PRESENT AT END OF YEAR. | | PER MILLION INHABITANTS. | |
|---|---|---|---|---|---|
| | | Blind. | Deaf Mutes. | Blind. | Deaf Mutes. |
| Italy | 1898 | 1070 | 2714 | 34 | 86 |
| Switzerland | 1898 | 131 | 639 | 42 | 207 |
| Austria | 1896 | 782 | 1416 | 31 | 57 |
| Russia | 1895 | 1132 | 1204 | 34 | 37 |
| England | 1896 | 1164 | 2584 | 38 | 84 |
| United States | 1896 | 3744 | 9832 | 51 | 135 |

# PHYSIOLOGIC CHEMISTRY.

By REID HUNT, M.D., and WALTER JONES, Ph.D.,
OF BALTIMORE, MD.

**Cystin.**—Mörner's[1] discovery of two isomeric forms of cystin as hydrolytic products of horn may furnish food for reflection to the artists who are accustomed to draw pictures of the proteid molecule without giving the sulphur atom any consideration. Interest was first given to the manner of the binding of sulphur in the proteids by the researches which were carried on in Liebig's laboratory, and which showed that by boiling proteids with alkalies a part of the sulphur can be split off in such a form as will precipitate lead sulphid from a solution of lead acetate, but that by far the greater part of the sulphur can not be so split off. It is, therefore, not surprising that Abel should find ethyl sulphid in the dog's urine, and that Aldrich should prove the presence of normal butyl mercaptan in the secretion of mephitis mephitica, which feeds only on proteid matter. It will be remembered also that from his researches on the oxidation of proteids with potassium permanganate Maly was led to the conclusion that in each proteid molecule there is one sulphur atom present as an SH group, which is oxidized by the permanganate to an $SO_3H$ group. So that if Kossel's assumption be true that the simplest proteids are the protamins which are sulphur-free, and that the sulphur present in ordinary proteids is to be found in conjugated side-chains, then our interest must be directed to the side-chain, for the occurrence of cystin in pathologic concretions is just as stern a fact whether we consider the sulphur of the cystin to be present originally as an essential part of the proteid molecule or not. The question whether the cystin of urinary calculi is to be considered as directly split off from the proteids of the body or as a result of synthetic processes can not be positively answered, but the work of Bauman and Preusse and of Bauman and Goldmann is to show that either cystin or a near relative of cystin occurs normally in slight amount in the urine, and that cystin is an intermediate product in the metabolism of sulphur which can be protected from further decomposition by the ingestion of halogen substitution products of benzene. The work of Mörner, however, seems to take the matter out of the field of argument. Pure defatted horn shavings were placed in a flask which was connected with a wash-bottle containing lead acetate. Enough dilute hydrochloric acid was added nearly to fill the flask, and without the introduction of any reducing agent the contents of the flask were heated on a water-bath at from 90° to 95° C. Only a trace of sulphureted hydrogen was given off, and among the products of hydrolysis were found two isomeric forms of cystin whose relative proportions depend upon the length of time during which the hydrolysis is carried on. One of these is ordinary hexagonal levorotatory cystin, and is formed in comparatively large quantity when the hydrolysis is carried on for a week. The substance gives analytic results which correspond to the composition of cystin, and when heated with an alkaline solution of lead acetate, it yields a copious precipitate of lead sulphid. By treatment with tin and hydrochloric acid it can be reduced to a substance which responds to all the color reactions for cystëin, and which by oxidation passes back again into cystin, from which the cystëin was prepared. Aside from optic properties and crystalline form the two cystins are scarcely to be distinguished from each other; indeed, there is no physical property of the substances which can serve for their separation, and they were to be obtained in comparative purity only by conducting the hydrolysis in such a manner that only one of the two products is formed, heating for 2 weeks having been found most favorable for the production of the needle form and 1 week for the hexagonal. In fact, while the needle-shaped crystals appeared to be an individual, and certainly have the composition of cystin, their specific rotation changes on recrystallization; so that all the preparations obtained were more or less contaminated with an isomeric but optically different cystin. From these and a countless number of qualitative reactions we can not escape the conclusion that the substance is in all respects identical with the hexagonal crystals of cystin which are to be found in urinary calculi. A check test, moreover, showed that the substance is not present as such in horn. On the other hand, when the hydrolysis of the horn shavings is allowed to proceed for 2 weeks, a large yield is obtained of a substance which has the composition of cystin, but which crystallizes in needles. Its chemical reactions and solubilities are practically those of hexagonal cystin; by reduction it is changed into cystëin, which is *apparently* identical with the corresponding substance obtained from hexagonal cystin, but which on oxidation, strangely enough, yields needles of cystin. In addition to these two forms of cystin, cystëin is also formed in considerable quantity in the hydrolysis of horn, and especially when the heating is prolonged. Moreover, the formation of cystëin is almost entirely prevented by conducting the hydrolysis in an atmosphere of hydrogen, from which Mörner concludes that cystin is the primary product from which cystëin is afterward formed by reduction. This work leaves little doubt that in the proteids of horn there is preformed a cystin group, or, at least, an atomic grouping which easily gives rise to cystin and which is accountable, in part at least, for the property which the proteids possess of giving off the so-called reduced sulphur when boiled with alkalies. This is one of the most interesting papers which has appeared since the YEAR-BOOK was last published, and even in the unusually large space which has been devoted to its abstraction it has been found impossible to mention many matters connected with what is one of the most interesting cases of geometric isomerism to be found in physiologic chemical literature.

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[1] Zeit. f. physiol. Chem., XXVIII, p. 595.

**Chemical Relationships of Colloid, Mucoid, and Amyloid Substances.**—The very remarkable work of Schmiedeberg showed that the chondroitin sulphuric acid of the cartilage is capable by hydrolysis of producing chondrosin, glycuronic acid, and glucosamin; and it has since been found that chondroitin sulphuric acid exists in some form of combination in the amyloid substance. Levene[1] now finds that tendomucin is a combination of a proteid with a nitrogenous ethereal sulphuric acid very similar to chondroitin sulphuric acid, since it yields chondrosin, and that submaxillary mucin, as well as colloid of a colloidal carcinoma, contains very similar or identical ethereal sulphuric acids in combination. The work points very clearly to the conclusion that colloid, mucoid, and amyloid substances are similarly constituted.

**Ovarial Mucoid.**—Leathes[2] has separated from the liquid of ovarian cysts a reducing substance which he names "paramucosin." This substance has the formula $C_{12}H_{13}NO_{10}$, and is considered to be a dihexosamin of some unknown carbohydrate.

**Normal Occurrence of Arsenic in Animals and Its Localization in Certain Organs.**—The statement has been made—without, however, adequate evidence—that arsenic occurs in all the organs of the body and in all articles of food. Gautier[3] finds most organs (liver, spleen, kidney, marrow, suprarenals, testicles, etc., and blood, urine, and feces) to be free of arsenic, but does find it constantly present in the thyroid of man and other animals; it also occurs in the thymus, brain, mammary glands, and milk, and, in traces, in bone, hair, and skin. The last-named structures seem to be the chief channel for the elimination of arsenic. One hundred grams of human thyroid yield on an average 0.75 milligram of arsenic; 1.3 grams of dried thyroid of the dog gave distinct reactions for arsenic. No arsenic could be detected in such articles of food as bread, eggs, and fish, but traces were found in potatoes and some other vegetables. Nucleins were prepared from the thyroid by peptic digestion; all the arsenic was found in these nucleins, the rest of the gland being entirely free of it. The arsenic probably exists in the nuclei in the form of "arsenical nucleins." The iodin was also found in the nucleins; there seems to be some connection between arsenic and iodin in the body, just as there is in the inorganic world. The medicolegal bearings of these observations are obvious; they show that the organs most commonly examined for arsenic in cases of poisoning are normally free of it, but they also show the necessity of examining each organ separately. The method employed by Gautier consisted essentially in destroying the organic matter with nitric and sulphuric acids, precipitating the arsenic with hydrogen sulphid, and applying the Marsh test; 0.0005 mg. in 100 parts of thyroid can be detected in this way. The usual method of destroying organic matter in toxicologic examinations (by means of potassium chlorate and hydrochloric acid) causes most of the arsenic to escape in a volatile form. A very delicate test for arsenic is said to be that of Abba, which consists in allowing Penicillium brevicaule to act upon the substance to be examined and observing the garlic-like odor produced. Scholtz [1] applied this method to the skin, hair, perspiration, and urine, and found that from 0.005 to 0.002 mg. arsenic could be detected. When testing the urine, it is advisable to first remove the odor of this liquid with animal charcoal, as it interferes somewhat with the test.

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[1] Arch. Neurol. and Psychopathol., II, p. 571.

[2] Arch. f. exper. Path. u. Pharmakol., XLIII, p. 245.

[3] Compt. rend., CXXIX, p. 929; CXXX, p. 284; and Bull. Acad. de méd., Dec. 5, 1899.

**Titanium in the Animal Organism.**—Baskerville [2] found human flesh to contain 0.0325% of titanic oxid; human bone contains but a trace, while 0.0195% was found in beef-bone.

**Iodin in Thymus and Thyroid.**—Gley found that the parathyroids of the dog and rabbit contain a relatively greater amount of iodin than do the thyroids of these animals; Mendel [3] finds the same in man, although in the individual examined the thyroid was abnormal and apparently contained an unusually small amount of iodin. Mendel, like some others, finds that the thyroids of new-born children contain no iodin; also that the thymus, when carefully isolated, is free of iodin. According to Charrin and Bourcet,[4] however, the presence or absence of iodin in the thyroid of the new-born child depends largely upon the condition of the mother's health. These authors found iodin (in amounts varying from 0.001 to 0.006% of the dried gland) in the thyroids of 14 children; some of the children had been born prematurely, others had lived a few weeks, but most of them had died during birth at term. Most of the mothers of these children were healthy. No iodin was found in the thyroids of 18 recently born children whose mothers were suffering from various pathologic, especially chronic, conditions.

**Uric Acid.**—Since the appearance of the Hopkins' method for the determination of uric acid in the urine so many criticisms have been offered that one must have a most thorough acquaintance with the literature to be able to make any intelligent use of the method. This method was accepted with eagerness by chemists who had used the exact but laborious method of Ludwig and Salkowski, but the results which were obtained by its use are now known to be incorrect, and in order to obtain results which are useful, the original method must be so far modified that it is scarcely recognizable as the method which Hopkins originally proposed. It will be remembered that Hopkins saturates a known volume of the urine with ammonium chlorid, filters off the precipitated ammonium urate, and washes the precipitate with a saturated solution of ammonium chlorid. The precipitate is then dissolved in water and the uric acid precipitated as such by the addition of hydrochloric acid, and after washing is determined by direct weighing or by titration with a standard solution of potassium permanganate. Hopkins himself knew that it is almost impossible completely to free the uric acid from chlorids, and afterward suggested washing the precipitated ammonium urate with a solution of ammonium sulphate until the washings are found to contain no chlorin—a process which, owing to the gelatinous character of the precipitate, requires several hours. Folin contributed a decided improvement when he found that under certain conditions the uric acid is completely precipitated by dissolving 20 grams of ammonium sulphate in 100 cc. of urine, and that the precipitated ammonium urate can be rapidly washed free from chlorin with a 20% solution of ammonium sulphate. It is perfectly familiar to all who have used this method that the end of the oxidation with permanganate is never sharp, and that with some specimens of pathologic urine one can never be sure that he has added an excess of the permanganate solution. This objection has been noted by Cazé,[1] Ritter,[2] and many others, and Jolles [3] has lately shown that both permanganate and iodin solutions are inadmissible in this connection. E. Mörner [4] objects to the use of Folin's modification, claiming that ammonium urate is completely precipitated from the urine by ammonium sulphate only under special conditions and when the urine is comparatively dilute. He claims that the following method gives exact results, and that the ammonium urate precipitated under the conditions as stated will be found easy to filter: Warm 150 cc. of urine to from 40° to 45° C., dissolve 30 grams of ammonium chlorid, and after standing for ½ to 1 hour, filter, and wash with 10% ammonium sulphate until the washings show no reaction for chlorin. Dissolve the precipitate in 2% caustic soda, warm until ammonia ceases to be given off, and determine the nitrogen in the residue by any convenient form of Kjeldahl's method. Jolles [5] finds that the Folin modification yields concordant results with solutions of pure uric acid, but that 1 cc. of a $\frac{1}{20}$ normal solution of potassium permanganate corresponds to 3.648 mg. of uric acid and not to 3.75 mg., as claimed by both Hopkins and Folin. [Ritter finds the constant 3.61.] On the assumption that 3.648 is the correct number, Jolles cites a number of comparative determinations to show that results obtained by Folin's modification are uniformly higher than those obtained by the laborious but exact Ludwig-Salkowski method. This leads Jolles to the assumption that there are substances in the urine, and especially in pathologic specimens, which are salted out by ammonium acetate, and which exert a reducing action on permanganate, and that the Folin modification must be entirely abandoned. He finds, however, that, contrary to the usually accepted view of the matter, the uric acid of the urine is quantitatively converted into urea by heating with a slight excess of potassium permanganate; thus,

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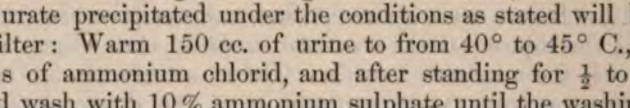
[1] Berl. klin. Woch., XXXVI, p. 913.

[2] Jour. Am. Chem. Soc., XXI, p. 1099.

[3] Am. Jour. Physiol., III, p. 285.

[4] Compt. rend. d. l'Acad. d. Sci., CXXX, p. 945.



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[1] Malay's Jahresbericht, 1895, p. 80.

[2] Zeit. f. physiol. Chem., XXI, p. 291.

[3] Zeit. f. physiol. Chem., XXIX, p. 193.

[4] Zeit. f. physiol. Chem., XXIX, p. 70.

[5] Zeit. f. physiol. Chem., XXIX, p. 223.

and proposes a new method, based on the estimation of the urea with a hypobromite solution. In from 50 cc. to 200 cc. of urine are dissolved from 5 to 20 grams of ammonium acetate and the solution is made faintly alkaline with ammonia. After standing for 2½ to 3 hours the ammonium urate is filtered off, washed with a saturated solution of ammonium carbonate, and warmed with very dilute caustic soda solution until the last trace of ammonia has been driven off. The liquid is acidified with sulphuric acid and treated at the boiling temperature with successive portions of permanganate until the pink color persists after boiling for ¼ hour. The excess of permanganate is reduced with oxalic acid and the urea is determined by Hufner's method. The process seems tedious and the urea estimation requires a special form of apparatus, so that it may be necessary to return to the Salkowski method.

His,[1] in collaboration with several others, makes an elaborate contribution to the subject of the **excretion of uric acid in gout.** He first calls attention to the necessity of continuing the observations in any given case over several (at least 6) days, since the uric acid excretion in gout undergoes the same variations, which are often very considerable, as those seen in health. The average daily excretion of uric acid in gout did not differ from that in health. For from 1 to 3 days before an acute attack, however, there was a decreased excretion; after the attack there was an increased excretion, which reached its maximum in from 1 to 5 days. During the attacks the excretion was about the same as during the intervals. No constant effects upon the uric acid excretion seemed to be produced by the use of various therapeutic agents; lithium carbonate seemed to diminish it slightly. Badt,[2] in a similar series of studies, found no decrease in the uric acid excretion during the attacks; on the contrary, it was usually increased. The author does not think these results are necessarily opposed to the view that the attack of gout is due to an accumulation of urates in the joints.

In a recent paper on the **chemistry of gout** Luff[3] emphasizes the relation between the gelatinous form of sodium biurate and this disease. The gelatinous form of this salt is easily soluble, while the crystalline form is almost insoluble; there seems to be an intimate relation between the gouty paroxysm and the deposition of this salt in the crystalline form in the tissues. If this change from the gelatinous to the crystalline form could be delayed, the gouty attack could be delayed. It was found that salts of sodium accelerated, while salts of potassium retarded, this conversion; lithium salts retarded the conversion to a slight extent. These results were in accord with the author's clinical experience. The conversion of the gelatinous sodium biurate into the crystallized form was much accelerated by a high alkalinity of the blood due to sodium bicarbonate. The alkalinity of the blood of gouty patients is always abnormally high; this is opposed to the prevailing view that gout is associated with an "acid dyscrasia." The latter view is, however, the purest assumption, and was disproved years ago by Klemperer. Luff found the alkalinity of the blood in gout to be nearly one-third greater than it is normally. The increased alkalinity probably determines the attack.

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[1] Deut. Arch. f. klin. Med., LXV, p. 156. [2] Zeit. f. klin. Med., XXXVII, p. 546.
[3] Lancet, 1900, I, p. 931.

Blumenthal [1] reports the results of experiments with **sidonal on uric acid formation.** Sidonal (prepared by Jaffé and Darmstädter, Berlin) is a compound of quinic acid and piperazin, both of which have already been recommended in the uric acid diathesis. In experiments on healthy men Blumenthal found the uric acid formation to be decreased from 30% to 50% by this new compound. Hippuric, instead of uric, acid was formed; the metabolism thus approached that of the herbivora. As uric acid requires 14,000 parts of cold water to dissolve it, while hippuric acid dissolves in 600 parts, the advantage of the change is obvious. The dose of sidonal is from 5 to 8 grams daily. Several clinicians claim to have had good results with this new drug.

Prout [2] discusses the occurrence of various **proteids in the urine,** the means of identifying them, and their clinical significance. Serum-albumin and serum-globulin are of especial importance on account of their connection with nephritis. Mucin is present in normal urine in small amount; it is increased in urethritis and cystitis, and so has some diagnostic value. The presence of albumose is of slight importance. Peptone usually indicates the formation somewhere of pus or the presence of cancerous conditions of the digestive tract.

Purdy [3] describes a clinical method for the **quantitative determination of albumin in the urine** which seems to be very simple and accurate and easily employed. The albumin is precipitated by acetic acid and potassium ferrocyanid in graduated tubes; the tubes are placed in a centrifuge making a certain number of revolutions a minute for 3 minutes. The amount of albumin is read off in bulk percentage, which, by the aid of a table, is converted into percentage by weight. The results obtained by this method were compared with those obtained by the gravimetric method, and the error was never greater than 0.01%.

Biffii [4] describes a very convenient method for the detection of **bile pigments in the urine.** The urine is made strongly acid with sulphuric acid, and barium chlorid is added; bile pigments, if present, are carried down with the precipitate. The latter is spread upon absorbent cotton and a crystal of potassium bichromate placed upon it; if bile pigments are present, a green ring, which becomes blue, and then red, forms around the crystal. Hammarsten [5] describes a modification of Huppert's test for bile pigments in the urine by which 1 part of pigment to 1,000,000 parts of urine can be detected; it has the great advantage that it can be applied to pathologic urines which contain other pigments in large amounts. A method for the quantitative determination of bilirubin in the urine by the action of iodin upon the pigment is described by Jolles.[6]

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[1] Berl. klin. Woch., XXXVII, p. 332.

[2] Phila. Med. Jour., V, p. 357.

[3] Jour. Am. Med. Assoc., XXXIII, p. 762.

[4] Centralbl. f. Harn. u. Sexualorgane, XI, p. 251.

[5] Skand. Arch. f. Physiol., IX, p. 313.

[6] Stzber. d. k. Akad. d. Wiss.; m.-nat. Cl., CVIII, Abth. II, b, p. 23.

**Excretion of Diamins in Cystinuria.**—Simon [1] reports a case of cystinuria in which cadaverin was found in both urine and feces; a review of the literature of cystinuria is given and the origin of cystin and the diamins discussed. Cammidge and Carrod [2] also report a case of cystinuria in which cadaverin was found in the urine and putrescin in the feces. The excretion of the diamins is very intermittent; for a period of 23 days in one instance none were found.

**Indican.**—The method of Wang [3] for the quantitative examination of urinary indican has been the subject of numerous criticisms. Wang treats the chloroform residue with a mixture of equal volumes of 96% alcohol, ether, and water for the purpose of removing a reddish-brown pigment which reduces permanganate, and would therefore be reckoned as indigo. The necessity for some such precaution had been noted by Obermayer,[4] who, while he believed the brown pigment to be an oxidation product of indigo, regarded the withdrawal of the substance as of smaller consideration than the leaving of other oxidizable compounds, which must always be present in the chloroform residue. As instances of such substances the following may be cited: Hippuric acid is broken up, yielding glycocoll, which is soluble in chloroform and subsequently converted into a sulphonic acid capable of reducing permanganate. So also phenol and the aromatic oxyacids, if originally present in the urine, would be found in the chloroform residue, and would exert a reducing action on the permanganate. Bouma,[5] however, claims that the "so-called" purification of the chloroform residue is not only superfluous, but actually introduces an error into the method, since the reddish-brown pigment is in all probability a mixture of depolymerized forms of indigo. He cites in support of this conclusion that pure indigo-blue is partly changed to indigo-red by boiling in chloroform solution. Wang is unable to verify this observation either with indigo prepared by Fritz's method or with indigo obtained from the urine by extracting the chloroform residue with his ether-alcohol-water mixture. He makes the very proper suggestion that the indigotin used by Bouma and prepared from commercial indigo is scarcely so reliable in such work as the pure crystalline substance prepared by oxidizing indigo-white. Wang also finds that the analytic results obtained are the same whether the chloroform be distilled in a vacuum, at ordinary pressure, or after the chloroform extract has been boiled for an hour—a result that can scarcely be brought into accord with the assumption that indigo is altered by boiling with chloroform. Wang also notes that the results are very seriously altered when the urine is allowed to stand a long time in contact with the mixture of ferric chlorid and hydrochloric acid, which might explain many apparent contradictions which are to be found in the literature.

There is still some difference of opinion as to whether any of the **oxalic acid of the urine** is derived from the oxalates of food. The older writers took it for granted that foods rich in oxalates (like spinach) caused an increase in the oxalates of the urine. Abeles several years ago denied this, and stated that there is no such thing as "alimentary oxaluria." Lommel[1] has recently published a paper which would seem to confirm the view of Abeles; he failed to find any increase of oxalic acid in the urine after the addition of oxalates to the food or after the consumption of spinach. Pierallini,[2] however, working under Salkowski's direction, obtained results which confirm the older views; experimenting on himself and upon convalescents, he found that both soluble and insoluble salts of oxalic acid were absorbed and passed into the urine as calcium oxalate. There was also an increase of the oxalates of the urine after the ingestion of food rich in oxalic acid (spinach). Hence all persons who have a tendency to excrete much oxalic acid in the urine should avoid food rich in oxalates. Salkowski[3] has investigated the origin of the oxalic acid of the urine. He concludes that the proteid is not the source of it; neither is it formed in pancreatic digestion or in the bacterial decomposition in the intestines. One source of it is uric acid; this is oxidized to oxalic acid. Nucleins and nucleoalbumins seem to be its ultimate source.

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[1] Am. Jour. Med. Sci., CXIX, p. 39.

[2] Jour. Path. and Bacteriol., VI, p. 327.

[3] Zeit. f. physiol. Chem., XXVII, p. 135.

[4] Zeit. f. physiol, Chem., XXVI, p. 427.

[5] Zeit. f. physiol. Chem., XXVII, p. 348.

Herringham[4] finds that the **toxicity of normal urine** and the amount of potassium salts contained in it vary together; the latter will account for nearly all the symptoms observed when urine is injected into the circulation of a rabbit. No difference exists between the morning and evening urine, as was supposed to be the case by Bouchard, and upon which his theory of sleeping and waking was founded. In fact, the theories of Bouchard upon the toxicity of the urine invariably fall to the ground when tested by experiment.

**Sources of Error in the Detection of Sugar in the Urine by Fehling's Solution.**—Eury[5] found that certain specimens of urine known to contain considerable quantities of sugar yielded no precipitate of cuprous oxid when boiled with Fehling's solution, although the latter was decolorized. This is due to the combination of creatin and similar bases with cuprous oxid to form soluble compounds; if the liquid is left to cool in the air, the upper layers assume a reddish-brown color, and a precipitate is finally formed, owing to the oxidation of the soluble copper compounds. The substances thus interfering with the detection of sugar may be removed by precipitation with mercury salts. Elliott[6] uses the following reagents for testing for sugar in the urine: No. 1.—Copper sulphate, c.p., 27 grains; glycerin, c.p., 3 drams; distilled water, 2½ drams; liquor potassa, q.s. ad 4 ounces. No. 2.—A saturated solution of tartaric acid in water. A dram of No. 1 is boiled and to it are added 3 drops of No. 2; the urine is added drop by drop until 8 drops have been added; 0.1% of sugar is detected in this manner. By adding ammonia these reagents may be used for quantitative determinations.

**Phenylhydrazin Test for Sugar.**—Neuman[7] gives the following simple method for detecting sugar in, for example, diabetic urine: Five cubic centimeters of the urine are mixed with 2 cc. of acetic acid previously saturated with sodium acetate; 2 drops of phenylhydrazin are added and the liquid is concentrated to 3 cc. Characteristic crystals of the osazone appear as the solution cools. Well-formed crystals were obtained when the solution contained but 0.01% of sugar. Coriat[1] has made investigations to determine whether there are substances in the urine which interfere with the phenylhydrazin test, and thinks that there are not if the urine is acid when the test is applied. According to Mayer,[2] glycuronic acid, in combination with which many drugs are excreted, forms a compound with the phenylhydrazin which has the same melting-point, appearance, and even elementary composition as the compound formed with sugar. Other tests, especially the fermentation test, must be made to distinguish these. A method for isolating this compound is described by Mayer and Neuberg.[3] It was shown that glycuronic acid may be obtained from normal urine, although in extremely small quantities.

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[1] Deut. Arch. f. klin. Med., LXIII, p. 599.

[2] Virchow's Archiv, CLX, p. 173.

[3] Berl. klin. Woch., XXXVII, p. 434.

[4] Jour. Path. and Bacteriol., VI, p. 158.

[5] Bull. de la Soc. Chim. (III), XXIII, p. 41.

[6] Medicine, VI, p. 17.

[7] Arch. f. (Anat. u.) Physiol., 1899, Suppl., p. 549.

Bergel and Blumenthal[4] describe a method for the **isolation of pentose** from the urine which consists essentially in treating the urine with barium hydroxid and then adding alcohol. The pentose forms a barium compound which is insoluble in alcohol; the pentose may be set free by treating this compound with $CO_2$ and the other sugars may be removed by fermentation. It has been impossible hitherto to obtain a pentose in pure form from the urine.

**Quinosol.** — Since the discovery of camphoglycuronic acid by Schmiedeberg and Meyer[5] it has been found that after the administration of any one of a large number of drugs there will be found in the urine a substance which, after boiling with dilute mineral acids, is capable of reducing Fehling's solution, and which causes the urine to show optic activity. These bodies, the paired glycuronic acids, have attracted attention because of a supposed possibility of confusing them with dextrose. They have, indeed, a peculiar interest for the physiologic chemist, but not for the reason stated, since there is at present scarcely a possibility of mistaking any one of these substances for grape-sugar. A most interesting member of this class has lately been discovered by Carl Brahm[6] in the urine of dogs and rabbits after the administration of quinosol. Quinosol is a substance that has recently found wide use as an antiseptic, an antipyretic, and a disinfectant. It is prepared by boiling an alcoholic solution of oxyquinolin with potassium pyrosulphate, and if the substance is oxyquinolinsulphonate of potassium, as claimed by its manufacturers, it should be formed according to the equation:

$$2C_9H_6NOH + K_2S_2O_7 = 2C_9H_6NOSO_3K + H_2O.$$

After giving the substance to rabbits in daily doses of 1 gram the urine was found on standing to deposit well-formed crystals, in some instances

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[1] Boston M. and S. Jour., CXLI, p. 518.

[2] Berl. klin. Woch., XXXVII, p. 5.

[3] Zeit. f. physiol. Chem., XXIX, p. 256.

[4] Arch. f. (Anat. u.) Physiol., 1900, p. 155.

[5] Zeit. f. physiol. Chem., III, p. 422.

[6] Zeit. f. physiol. Chem., XXVIII, p. 439.

½ cm. in length, and in greater quantity when the animal was fed with food which is acid and as free from water as possible. With dogs, and with rabbits when no precautions were taken to secure a concentrated urine, no such deposition of crystals was observed, but in all cases the urine was found to be levorotatory, to be capable of holding large quantities of copper oxid in solution, and to reduce copper solutions after boiling with dilute mineral acids. Whether crystals are deposited or not, the acid can be isolated from the urine by precipitating successively with neutral and basic lead acetate and decomposing the second precipitate with sulphureted hydrogen. The filtrate from the lead sulphid, when sufficiently concentrated, deposits the glycuronic acid in large transparent pyramids. From the free acid the potassium salt was prepared, analyzed, and found to be in accordance with the formula $C_{15}H_{14}NO_7K$. The free acid, moreover, was boiled with dilute sulphuric acid and both glycuronic acid and oxyquinolin were found among the hydrolytic products. These experiments leave no doubt that the substance is oxyquinolinglycuronic acid, and it is difficult to understand how this substance can owe its origin to the administration of quinosol if quinosol indeed be oxyquinolinsulphonate of potassium. All the evidence at our disposal goes to show that the sulphonic acids are not broken up in the body. Salkowski [1] has shown that the potassium salt of metaphenol sulphonic acid appears unaltered in the urine, and the same has been found by Rabuteau [2] to be true of the corresponding para compound. Nor is it probable, as claimed by some, that quinosol is an ethereal sulphate, since these substances also have been found to pass quantitatively into the urine. Brahm, moreover, makes the following observations: (1) From a faintly alkaline solution of quinosol oxyquinolin can be extracted with chloroform; (2) from an aqueous solution of quinosol barium chlorid will precipitate the sulphur quantitatively as barium sulphate; (3) after treating quinosol with cold absolute alcohol the residue may be identified as potassium sulphate; (4) oxyquinolin obtained by hydrolysis of the crystalline substance found in the urine after the administration of quinosol will produce the same glycuronic acid when administered to an animal. It can, therefore, scarcely be doubted that quinosol is a mixture of potassium sulphate and oxyquinolin sulphate, and that any pharmaceutic value which the drug possesses should be ascribed to one or both of these substances.

Schumacher and Jung [3] describe a method by which very small quantities of **mercury in the urine** may be determined fairly accurately. The method consists essentially in destroying the organic matter of the urine with potassium chlorate and hydrochloric acid, reducing the mercuric chlorid thus formed with stannous chlorid, and filtering through gilt asbestos mixed with granules of gold. The gilt asbestos is prepared by steeping asbestos in gold chlorid and then reducing with hydrogen. Such a filter arrests every particle of mercury; when it is heated, the mercury is expelled and the loss of weight gives the weight of the mercury. Farup [1] modifies this method in one or two details and finds that half a milligram of mercury can be detected in a liter or more of normal or pathologic urine. Eschbaum [2] has also described a clinical method for the detection of mercury in the urine; the mercury is collected on bright copper foil, from which it is driven off by heat and collected on silver. The increase in weight of the latter is the weight of the mercury. Eschbaum found mercury in the urine 48 hours after the first application of a mercurial ointment; it was also found 6 weeks after the last application.

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[1] Pflüger's Arch., 4, 91.

[2] Maly's Jahresbericht, 1881, 195.

[3] Arch. f. exper. Path. u. Pharmakol., XLII, p. 138; also Lancet, I, 1900, p. 92.

**Glycosuria Following Thyroid Feeding.**—The observation that thyroid feeding sometimes causes glycosuria led Porges [3] to make a number of experiments upon a dog on this subject. He found the urine to contain a fermentable levorotatory reducing substance during and for some time after the administration of thyroid; sometimes the glycosuria was 0.5%. If the cane-sugar of the diet were replaced by an equal amount of dextrose, then the urine became dextrorotatory. These experiments furnish a new illustration of the injurious effects which may follow thyroid feeding.

**Glycolytic Function of the Pancreas.**—The diabetes associated with lesions of the pancreas has been thought to be due to the diminution or disappearance from this organ of a sugar-destroying ferment, and a certain amount of evidence has been brought forward to show that such a ferment is normally present in the pancreas. Umber,[4] however, thinks that these results are due to bacterial action; he finds that when strict antiseptic precautions are taken in preparing extracts of the gland, the pancreas has no greater sugar-destroying power than other tissues. There seemed also to be no difference between the glycolytic power of the blood entering and that leaving the pancreas—a further indication that this organ does not secrete a ferment into the blood. Pierallini [5] has examined the pancreas of patients dead of various diseases for such a ferment; it seemed to be present in some, but not in others. The material was not, however, aseptic. Urine, whether obtained from healthy individuals or from those suffering from diabetes or other diseases, showed no glycolytic power.

**The Lactase of the Pancreas.**—According to Weinland,[6] there is an enzyme, lactase, in the pancreas which converts lactose into dextrose and galactose; Fischer and Nickel had failed to find such an enzyme. The amount of lactase is increased by a milk diet. The author also found that lactose may be split up into dextrose and galactose by an organic (citric) acid; no intermediate products were obtained.

**Formation of Glycogen from Fat.**—Bouchard and Desgrez [7] find that while the administration of fat to a starving animal does not increase the glycogen of the liver, that of the muscle is increased; this is ascribed to the incomplete oxidation of the fat. The sugar of the blood

[1] Arch. f. exper. Path. u. Pharmakol., XLIV, p 272.

[2] Deut. med. Woch., XXVI, p. 52.

[3] Berl. klin. Woch., XXXVII, p. 300.

[4] Zeit. f. klin. Med., XXXIX, p. 13.

[5] Zeit. f. klin. Med., XXXIX, p 26.

[6] Zeit. f. Biol., XXXVIII, p. 607.

[7] Compt. rend. d. l'Acad. d. Sci., CXXX, p. 816.

that leaves the liver is considered another source of the muscle glycogen. On oxidation, muscular glycogen is stated to pass into the condition of lactic acid, and not into that of sugar. Rosenquist[1] thinks that in severe cases of diabetes mellitus fat may be a source of sugar, as is claimed to be the case by Seegen and Rumpf. Lüthje[2] reports numerous observations on metabolism during diabetes; special attention was given to the relation between the sugar excreted and the kind of proteid ingested. Casein and pancreas caused a greater excretion of sugar than did beef, and the latter more than did egg-albumen. No evidence was found for the formation of sugar from fat. Sternberg[3] endeavors to show that diabetic coma is not caused by β-oxybutyric acid or its decomposition products (diacetic acid and acetone), but by a mother-substance of oxybutyric acid, perhaps β-amidobutyric acid. The latter in very small doses causes symptoms very similar to those of diabetic coma. Grube[4] also opposes the theory that oxybutyric acid is the cause of diabetic coma, and reports 12 experiments upon cats in which he succeeded in producing a condition very similar to diabetic coma by the intravenous injection of β-amidobutyric acid.

**A process for the preparation and estimation of glycogen** is described by Gautier[5]; the method is based on the fact that the nitrogenous substances with which glycogen is associated are completely precipitated by mercuric acetate in neutral solution. The glycogen is purified by repeated solution in water and reprecipitation with alcohol. Glycogen from the human liver yields, when heated with mineral acids, products having a higher reducing power than glucose; their reducing power is also greater than that of the products obtained from the glycogen of the rabbit's liver. From the foregoing and other considerations it is concluded that different varieties of glycogen are contained in the same organs of different animals and in various organs of the same animal. The liver glycogen is increased during pregnancy, according to Charrin and Guillemont[6]; this increase is much greater if glucose is injected into the animals. De Sinety[7] thinks that this increase of glycogen is connected with the function of lactation soon to be developed, whereas Charrin and Guillemont[8] explain it by supposing that less sugar is consumed owing to a lowering of general metabolism during pregnancy.

**Origin of Acetone in the Urine.**—One suggestion as to the cause of the appearance of acetone in the urine is that it is due to the excessive formation of acid in the organism. Lüthje,[9] however, failed to find any constant increase in the excretion of acetone as a result of strychnin convulsions (which cause an increased production of lactic acid), or after epileptic attacks (in which the formation of lactic acid is also probably increased), or after the administration of calomel (to increase the activity of the intestines). Waldvogel[10] brings forward arguments and experiments to show that the acetone of the urine arises from neither the proteids nor carbohydrates, but from the fats. Since fats injected subcutaneously do not increase the excretion of acetone, while those given by the mouth do, it is considered that processes in the intestine play an important part in its formation. Oppenheimer[1] describes a method for the detection of acetone in the urine and blood by the use of acid mercury sulphate; Denigès,[2] however, claims to have already described this method.

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[1] Berl. klin. Woch., XXXVI, p. 612.
[2] Zeit. f. klin Med., XXXIX, p. 397.
[3] Zeit. f. klin. Med., XXXVIII, p. 65.
[4] Brit. Med. Jour., 1900, p. 577.
[5] Compt. rend. d. l'Acad. d. Sci., CXXIX, p. 701.
[6] Compt. rend. de la Soc. de Biol., LII, p. 211.
[7] Ibid., p. 228.
[8] Ibid., p. 247.
[9] Centralbl. f. innere Med., XX, p. 969.
[10] Centralbl. f. innere Med., XX, p. 729.

**Lactic Acid in the Animal Organism.**—According to Morishima,[3] sarcolactic acid is a constant constituent of the fresh normal liver, kidney, and blood. The lactic acid of the liver undergoes a postmortem increase, probably at the expense of the glycogen; this acid is, however, fermentation lactic acid. Arsenic causes an increase of sarcolactic acid in the body; glycogen disappears from the liver at the same time, but the formation of the acid seems to be independent of this.

**Ferments.**—The work of Osborne[4] on the inverting ferment of yeast will be specially interesting to those who believe that there is no fundamental difference in the modes of action of organized and unorganized ferments. This investigator has obtained from the yeast by somewhat different methods a number of preparations which exhibit strong inverting power and whose analyses agree fairly well with one another when we take into consideration the physical properties of the substance and the consequent difficulty which must attend its purification. The yeast was rubbed to a paste with alcohol and allowed to stand for the purpose of coagulating the proteids, and after removing the alcoholic extract the residue was digested for a long time with warm water and filtered. This filtrate was treated with alcohol, and an aqueous extract of the precipitate thus obtained was treated with ammonia and a quantity of magnesia mixture sufficient for the precipitation of all the phosphoric acid. The solution was then dialyzed against running water, advantage being taken of Siegfried's arrangement,[5] by which the liquid within the parchment vessel, as well as the water on the outside, is kept continually in motion. The dialyzed solution was concentrated at a low temperature, precipitated with alcohol and ether, and the precipitate dried over sulphuric acid. The preparation thus obtained is extremely active, and is shown to be free from proteids by the failure of its aqueous solution to produce a precipitate either with mercuric chlorid or Mayer's reagent. The author notes the similarity in composition of his preparation to that of chitin and Lücke's hyalin:

| | CHITIN. | INVERTIN PREPARATION. | HYALIN. |
|---|---|---|---|
| C . . . . | 46.35 | 44.69 | 45.30 |
| H . . . . | 6.44 | 6.51 | 6.50 |
| N . . . . | 6.01 | 6.10 | 5.20 |

While such analytic agreements are so common that they usually deserve little attention, the following observation shows that the agree-ment may in this instance be something more than a coincidence. A solution of chitin when boiled with hydrochloric acid yields a dark brown humus substance and glycuronic acid. So also a solution of the invertin preparation when similarly treated deposits a black pigment, and the solution forms yellow crystalline needles with phenylhydrazin which melt at 194° C. and contain the same percentage of nitrogen as glucosazone.

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[1] Berl. klin. Woch., XXXVI, p. 828.

[2] Ibid., XXXVII, p. 43.

[3] Arch. f. exper. Path. u. Pharmakol., XLIII, p. 215.

[4] Zeit. f. physiol. Chem., XXVIII, p. 399.

[5] Ber. d. d. chim. Ges., XXXI, p. 1825.

**Formation of HCl in the Stomach.**—One theory of the formation of the HCl of the gastric juice is that there is an interchange between the H ions of the acid carbonates and phosphates of the blood and the Na ions of the NaCl in the stomach. Wesener [1] disproves this theory by showing that when the mucous membrane of the stomach is irritated, free HCl appears whether any NaCl is present or not; also that no HCl is formed when NaCl is present unless the mucous membrane is stimulated. The acid is considered to be the result of cell activity. Cohnheim and Krieger [2] describe a method for the determination of the combined HCl of the gastric juice. The total acidity of the gastric juice is first determined, phenolphthalein being used as the indicator. Phosphotungstate of calcium is then added; the proteid is precipitated, and the HCl in combination with it unites with the calcium. The acidity is again determined; the difference between the figures obtained in the two determinations gives the amount of acid combined with the proteids. The following method (a modification of the Töpfer method) is recommended by Hewes [3] for the quantitative determination of the acidity of the gastric contents: To 5 cc. of the well-shaken but unfiltered stomach-contents add a drop or two of a $\frac{1}{2}\%$ solution in alcohol of dimethyl-amido-azobenzol, and then a sufficient quantity of a decinormal solution of sodium hydroxid to just convert the red color into an orange or bright yellow, the end-reaction being perhaps facilitated by adding also a drop of tropeolin; continue to add the decinormal solution of sodium hydroxid until a drop of the mixture fails to color Congo-red paper; adding a few drops of a 1% alcoholic phenolphthalein solution, continue to add the decinormal sodium hydrate solution until a deep red color of maximum intensity appears. The reading from the beginning (*a*) up to the end of the first procedure gives the total free HCl; (*b*) to the end of the second procedure, the total free acids plus acid salts; (*c*) to the end of the third procedure, the total acidity. (*d*) By subtracting (*a*) from (*b*) we have a quantitative estimate of the total organic acids plus acid salts; (*e*) by subtracting (*b*) from (*c*) we obtain the total combined acids (the total combined HCl, when free HCl is present). (*f*) By adding (*a*) and (*e*) we get the secreted HCl. The analysis can be accomplished in a few minutes, requires but a few reagents, and, from the foregoing findings, nearly everything of importance about the acid secretion of the stomach is obtainable, while the simplicity of the method renders it capable of application as a routine means of diagnosis in practice. **Parachymosin** is the name given by Bang [4] to a rennet ferment which he has found in the stomach of pigs and man; it differs in some respects from *chymosin*, as the ordinary rennet ferment is termed here.

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[1] Pflüger's Arch., LXXVII, p. 483.

[2] Münch. med. Woch., XLVII. p. 381.

[3] Boston M. and S. Jour., CXLII, p. 9.

[4] Pflüger's Arch., LXXIX, p 425.

**Digestibility of the Casein of Cow's and Human Milk.**—It has been stated that a larger percentage of phosphorus in organic combination appears in the feces of children when fed on cow's milk than when they receive human milk: that is, the casein of the former is not so readily digested as that of the latter. Müller,[1] however, finds this not to be the case; the casein of one kind of milk is as completely digested as that of the other, and the absorption is complete in each case. The digestion of casein in adults is as complete as in children. According to Kobrak,[2] the caseinogen of human milk differs from that of cow's milk chiefly in its small degree of acidity; he thinks that this may be due to an admixture of some basic proteid-like material, possibly histon or protamin. Lührig[3] has tested the digestibility of two lard substitutes; one consisted of 2 parts of tallow and 3 parts of cotton-seed oil, while the other was made of equal parts of the first and of lard. The digestibility of these by the human intestine was about the same as that of genuine lard; approximately 95% was absorbed in each case.

**Absorption of Iodin Oils.**—Rösel[4] finds that iodin of iodized fats is more quickly absorbed than is that of inorganic compounds. From 43% to 79% of the iodin was recovered from the urine in from 50 to 58 hours.

Pflüger[5] contributes another polemic article on the question as to the **formation of fat** from proteid, in which he discusses results recently obtained by Cremer. Cremer found that some of the carbon of meat fed to cats was retained in the body; he thought that a part of this was retained as fat. Pflüger holds that this carbon may have been held back in leucin, tyrosin, glycogen, etc., or in the undigested and putrid meat remaining in the intestinal tract. Pflüger points out what he considers to be other sources of error in Cremer's work, and maintains that there is as yet no evidence for the formation of fat from proteid. Lindemann[6] compares the fat of degenerated heart muscle with that from normal hearts and from other parts of the body, and finds that it differs from these; the degeneration fat has a high saponification number and a large percentage of volatile fatty acids, thus resembling butter fat. These results support the view that the fat of the degenerated organ arises in the cells, and is not simply transported thither from other organs. Benedict and Osterberg[7] give the results of some analyses of human fat. They find that the composition of this fat is remarkably constant; the average of 24 determinations gave: C, 76.08%; H, 11.78%. The heat of combustion averaged 9.523 large calories per gram.

**Metabolism During Poisoning by Pulegone.**—Pulegone, the active constituent of the ethereal oil of *Mentha pulegium*, causes, when injected subcutaneously, great fatty degeneration of various organs.

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[1] Zeit. f. Biol., XXXIX, p. 451.

[2] Pflüger's Arch., LXXX, p. 69.

[3] Zeit. f. Nähr. u. Genussm., III, p. 73.

[4] Pflüger's Arch., LXXVII, p. 22.

[5] Pflüger's Arch., LXXVII, p 521.

[6] Zeit. f. Biol., XXXVIII, p. 405.

[7] Am. Jour. Physiol., IV, p. 69.

Lindemann[1] has investigated the metabolism of dogs poisoned with this substance, and finds the destructive metabolism of proteid much increased; the excretion of $CO_2$ was but little altered. It was not determined whether the total amount of fat in the body was increased or not; along with the increased production there was an increased consumption of fat. According to Santesson,[2] benzol belongs to the poisons causing fatty degenerations. Rabbits poisoned with this substance showed fatty degeneration of heart and kidney, etc.; fat emboli in the lungs were also found. There seemed to be, in addition, a solution of body-fat and an accumulation of it in the kidney. These changes are caused by the benzol itself, not by phenol, which might have been formed from the benzol.

**Absorption of Iron.**—The experiments of Abderhalden[3] bring additional support to the view, now generally accepted, that inorganic iron is absorbed. The absorption of ferric chlorid by the intestines was studied by microchemical methods, chiefly in rats. The duodenum was found to be the principal seat of absorption; iron is excreted by the large intestine. Abderhalden's experiments differ from others chiefly in that he used very small amounts of iron. In a second paper[4] on this subject the author states that animals on their normal diet assimilate more iron than those kept on a diet poor in iron to which inorganic iron salts, hemoglobin, or hematin has been added.

In order to test the correctness of Asher's view that the principal **path for the absorption of proteids** is the lymphatic system, Levene and Levin[5] performed a series of experiments upon dogs in which carefully prepared iodoproteid was ingested, thus adopting conditions which permitted a clear differentiation of the proteid ingested from the tissue proteids. The ingestions of the proteids were made variously in the large intestine, the colon, the small intestine, and along the entire digestive tract, and the lymph was collected from the thoracic duct from 1 to 7 hours after the ingestion; yet in no instance could iodoproteid be shown in the lymph. The experiments are very conclusive in showing that the lymph is not a path of absorption of the proteids. Munk and Lewandowsky[6] have investigated the fate of proteids after their introduction into the circulation, and find that only those which have been profoundly altered, like casein, are not utilized; others, like caseinogen, egg-albumen, acid, and alkali albumin, are, contrary to the opinion of many, utilized to a greater or less extent.

**Sandy Matter from the Human Intestine.**—Thomson and Ferguson[7] examined sandy matter passed with the feces by a woman suffering with catarrh and dilation of the stomach. The sand was readily soluble in dilute acids, and consisted of 71.5% of inorganic and 28.5% of organic matter. The former consisted largely (87.3%) of tricalcium phosphate; calcium carbonate (11.7%) and an insoluble residue (largely silica) made up the remainder. Among the organic materials particular attention was given to a brown pigment, the exact nature of which was not determined; it is possibly an intermediate substance between bile pigment and stercobilin.

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[1] Zeit. f. Biol., XXXIX, p. 1.

[2] Skand. Arch. f. Physiol., X, p. 1.

[3] Zeit. f. Biol., XXXIX, p. 113.

[4] Zeit. f. Biol., XXXIX, p. 487.

[5] Arch. Neurol. and Psychopathol., II, p. 553.

[6] Arch. f. (Anat. u.) Physiol., 1899, Suppl., p. 73.

[7] Jour. Path. and Bacteriol., VI, p. 334.

**Two methods for the estimation of phosphorus in organic matter** are described in which the organic matter is destroyed without the use of the sealed tube. Marie [1] destroys the organic matter with potassium permanganate and $HNO_3$; the phosphorus is then determined in the usual way. The method of Neumann [2] is applicable to liquids (milk, urine, etc.) as well as to solids; it consists in the use of $H_2SO_4$ and $HNO_3$ for destroying the organic matter. By working carefully and with acids of the proper strength the organic matter may be destroyed without any reduction to carbon. The liquid is then titrated with a molybdic solution.

**Metabolism of Phosphorus.**—Leipziger [3] confirms the results obtained by Steinitz that feeding with proteids containing phosphorus yields better results so far as the putting on of phosphorus is concerned than feeding with phosphorus-free proteids to which inorganic phosphates have been added. Leipziger used edestrin; Steinitz myosin—both of which are phosphorus-free proteids. The experiments were made upon dogs. According to Paton, Dunlop, and Aitchinson,[4] the intestines and not the kidneys are the chief seat for the excretion of the phosphorus of the food; this is especially true of the herbivora. Only about 1 % of the phosphorus of the food was found in the urine of the goat; a somewhat larger amount was found in that of the dog upon a vegetable diet. When sodium phosphate was given subcutaneously, very little of the phosphorus appeared in the urine. During lactation the amount of phosphorus in the goat's feces and in the dog's urine was diminished to meet the requirements of milk formation. Goat's milk contains a high percentage of phosphorus, but less of it is in organic combination than in human or cow's milk. The administration of the soluble calcium glycerophosphate causes no rise in the phosphorus of the urine of the dog or in the urine or milk of the goat.

**Metabolism of Nucleins.**—Milroy and Malcolm [5] found in a case of lymphatic leuckocythemia that the excretion of phosphoric oxid was diminished both absolutely and relatively to the nitrogen excreted; the excretion of uric acid and alloxuric bases was hardly affected. In a case of medullary leukocythemia when the number of leukocytes was rapidly falling the excretion of phosphoric oxid was not diminished, while the alloxuric excretion underwent great variations. The greater part of the paper is devoted to a michrochemical study of the granules of the leukocytes and the action upon them of nucleïc acid. According to Loewi,[6] the quantity of phosphoric and uric acids in the urine depends largely upon the amount of nuclein in the food. Allantoin was not found in the urine (man) after the ingestion of large amounts of pancreas or thymus; it is not, therefore, an end-product of the metabolism of nuclein. As has already been shown by Minkowski, very little allantoin is found in the urine after its ingestion.

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[1] Bull. de la Soc. Chim. (III), XXIII, p. 44.
[2] Arch. f. (Anat. u.) Physiol., 1900, p. 159.
[3] Pflüger's Arch., LXXVIII, p. 402.
[4] Jour. Physiol., XXV, p, 212.
[5] Jour. Physiol., XXV, p. 105.
[6] Arch. f. exper. Path. u. Pharmakol., XLIV, p. 1.

**Xanthin Bases in the Feces.**—Patién[1] has shown that xanthin bases are excreted in the feces to a considerable extent when an animal is kept upon a milk diet. Inasmuch as these bases can not be obtained from milk, those in the feces must arise from the body itself, probably from the alimentary tract. Parker[2] obtains similar results. On a diet free from nucleins the day's feces contain about 30 mg. of xanthin bases—approximately the same as the urine; this amount could be nearly doubled by giving a mixed diet or one containing nucleins.

**N-Metabolism of the Cat.**—Mendel and Brown[3] record some observations upon the occurrence of certain substances in the cat's urine. Kynurenic acid, which is present in the urine of the dog, is absent from that of the cat and of man. The uric acid output per kilo body-weight in the cat is about the same as in man and the dog, and is increased by feeding thymus and pancreas. Allantoin excretion was observed after thymus and pancreas feeding. Creatin is present in the cat's urine in noticeable quantity.

Most of the experiments upon the **influence of boric acid and formalin upon metabolism** have been performed upon adults, and seem to show that these substances, in small amounts, do not have any marked influence. Annett[4] found very different results in experiments upon young animals; no kitten lived longer than 4 weeks when fed on milk containing 40 grains of boric acid to the gallon (about the amount ordinarily used to preserve milk). Similar results were obtained with formalin in strengths of 1 part to 12,500 or 50,000; many of the kittens died, and those which survived grew slowly and were in a very bad condition. These experiments show the danger which attends the use of these substances as preservatives for the milk intended for young children.

**Influence of Water upon Metabolism.**—Straub[5] finds that removal of $H_2O$ from the food increases proteid catabolism; the excretion of both phosphorus and nitrogen is increased. There is no increased consumption of fat, as assumed by Oertel. The blood pressure is little affected unless pathologic symptoms are produced. The effect of NaCl in producing a slight increase of catabolism is attributed to the loss of $H_2O$ caused by the diuretic action of the salt. Very large quantities of $H_2O$ have, according to Neumann,[6] no influence on metabolism other than a flushing out of the products of proteid catabolism; there was no increase of proteid metabolism.

**Oxidation of Carbon Monoxid by the Body.**—A number of recent writers state that CO is oxidized or otherwise destroyed in the living body; Haldane[7] has obtained opposite results. Mice were placed in a bell-jar containing a certain percentage of the gas; the amount of CO was found unaltered even after 29 hours.

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[1] Skand. Arch. f. Physiol., IX, p. 412.

[2] Jour. Physiol., IV. p. 83.

[3] Am. Jour. Physiol., III, p. 261.

[4] Lancet, 1899, p. 1282.

[5] Zeit. f. Biol., XXXVIII, p. 537.

[6] Arch. f. Hyg., XXXVI, p. 248.

[7] Jour. Physiol., XXV, p. 225.

Stephens [1] draws the following general conclusions from experiments on **snake toxins and toxic serums**: (1) An antitoxic serum can act on toxins other than, but allied to, that used in the preparation of the serum; (2) the hemolytic constituents of snake toxins, and hence snake toxins as a class, are not identical; (3) 0.5 cc. of Calmette's antivenom has very little action against a minimum lethal dose of daboia toxin; (4) the antihemolytic properties of antivenomous serums must be increased in order to afford any protective serum, for instance, against pseudechis or daboia toxin. A general article on the chemistry and toxicology of snake-poisoning, with a review of some of the recent opinions on the relation of toxins and antitoxins, is contributed by Brown.[2] Faust [3] has studied the poisons of the skin of the salamander; he finds two alkaloids, samandarin and samandaridin, which form crystalline sulphates and yield isoquinolin on distillation with zinc dust. From 0.7 to 0.9 mg. of samandarin per kilo body-weight was fatal to dogs; the symptoms were similar to those of rabies. Samandaridin is less poisonous. The poison occurring in the skin of *Spelerpes fuscus*, a small Italian salamander, has been investigated by Benedicenti and Polledro; [4] they think it is an acid, perhaps a derivative of one of the cyanacetic acids. The poison is a powerful local irritant, abolishes reflexes, and kills by paralyzing respiration and heart. Red blood-corpuscles are destroyed by the poison.

**Analysis of the Blood.**—Jellinek and Schiffer [5] find that ordinary healthy blood has a specific gravity of 1.060 to 1.065, leaves 22.26% to 23.94% of dry residue, and contains 0.0431% to 0.0527% of iron. The various constants of the blood of healthy beings were found to maintain a certain relationship, and in pathologic cases the specific gravity and percentage of dry residue seemed also to be connected; the iron, however, varied independently.

Burmin [6] gives, in the form of tables, the results of observations upon the **alkalinity of the blood** in a large number of conditions, and the effect upon it of drinking mineral waters; the latter, in moderate amounts, cause an increase in the alkalinity only after several days' use.

Panzer [7] reports the analyses of 2 specimens of **cerebrospinal fluid** of children. One specimen had a specific gravity of 1.0086; the other, 1.00917. The latter contained a hexose. Both were faintly alkaline and contained cholesterin, neutral fats, and soaps, but albumoses and urea were not found.

Vertun [8] examined a specimen of **hydrocele fluid**; he found traces of NaCl and phosphates and 0.44% of proteid, mostly serum-albumin. Albumose and spermin were absent; spermatozoa were present and were very active—an observation which shows that albumoses and spermin

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[1] Jour. Path. and Bacteriol., VI, p 273.

[2] Johns Hopkins Hosp. Bull., X, p. 221.

[3] Arch. f. exper. Path. u. Pharmakol., XLIII, p. 83.

[4] Arch. ital. d. Biol., XXXII, p. 135.

[5] Wien. klin. Woch., XII, p. 802.

[6] Zeit. f. klin. Med., XXXIX, p. 365.

[7] Wien. klin. Woch., XXII, p. 805.

[8] Centralbl. f. d. med. Wiss., XXXVII, p. 529.

(and probably other secretion products of the accessory reproductive glands) are not necessary stimulants to the movements of the spermatozoa.

The percentage of **urea in the blood** in a number of pathologic conditions has been determined by Herter [1] and its relation to the condition known as uremia discussed. The conclusion reached is that a patient is in danger of developing unequivocal symptoms of uremia whenever the urea-content of the blood exceeds 0.2%, and that a fatal issue will almost certainly occur when the percentage exceeds 0.3%. On the other hand, in some conditions classed as uremias the percentage of urea may be below normal, and probably has little influence upon the individual's condition.

Bing [2] has published an important paper on the **reducing substances of the blood.** The reducing substances soluble in water were estimated as dextrose; those soluble in ether as jecorin—a reducing body known to be present in the blood in considerable quantities. The blood of animals in which the diabetic puncture was made showed a considerable increase in the amount of jecorin in the blood, while the sugar showed no constant increase. Extirpation of the pancreas was followed by an increase of both jecorin and sugar. Jecorin is regarded by the author as a combination of sugar and lecithin; he thinks that they unite directly in alcoholic solution. According to this view, jecorin is an artificial product; the form in which the sugar exists in the blood is unknown. Contrary to Seegen, Bing found no great difference between the amount of sugar in the hepatic and other veins; Seegen's results are attributed to the stasis produced in the vessels of the liver.

**Milky Serous Effusions.**—Shaw [3] describes an interesting case of chylous ascites and hydrothorax, and compares it with cases described by others. The cause of the affection was a mass of lymphadenomatous glands pressing on the thoracic duct. There were no visible fat particles in the fluid and it did not become clear when shaken with ether, but became jelly-like. The urine also became gelatinous when shaken with ether even when the albumin it contained had been removed by boiling. The ascitic fluid contained 4% of solids, 2.2% of proteids, 0.52% of fat, and 0.7% of ash. Stcherbalscheff [4] has described a similar case of milky serous effusion; he thinks that the opacity of the liquid is connected in some way with the presence of a proteid substance, but is unable to explain the relation.

**Embryochemical Studies.**—Levene [5] gives an account of the first of a series of researches which will have for their object the study of the changes in the chemical distribution of nitrogen and phosphorus in the fertilized egg as development proceeds. The material used was the egg of the codfish, and this was examined in 4 stages: unfertilized, 24 hours after fertilization, 11 days after fertilization, and 20 days after fertilization. The analytic results obtained furnish the data for calculating any changes that occur in the quantities of amido-acids, proteids, nuclein bases, or nucleoproteids, as the development of the egg proceeds. While the author feels that it would be premature to draw any broad conclusions from the data thus far obtained, the results point strongly to the conclusion that in the course of development the processes of synthesis are preceded by those of decomposition. In the first stage after fertilization the proteids diminish in quantity and basic nitrogenous substances are formed at their expense. Later the basic substances decrease in quantity and the proteids increase. The combined proteids (nucleoproteids) and mineral substances greatly increase in the course of growth.

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[1] Contrib. to Sci. of Med., ded. to W. H. Welch, p. 69.

[2] Skand. Arch. f. Physiol., IX, p. 336.

[3] Jour. Path. and Bacteriol., VI, p. 339.

[4] Physiologiste Russe, I, p. 267.

[5] Arch. Neurol. and Psychopathol., II, p. 557.

Hugounenq[1] determined the proportion of potassium and sodium in the ash of fetuses of different ages. As growth takes place both potassium and sodium increase in amount, but the latter increases more rapidly on account of the richness of cartilage in NaCl. The potassium increases more rapidly in well-nourished subjects than in cases of malnutrition; this is because potassium is so abundant in red blood-corpuscles. Hugounenq[2] and Camerer[3] report analyses of the ash of new-born children. The most interesting part of the work consists in the comparison of the ash of the child and that of the milk of the mother. Bunge had made similar comparisons, using lower animals, and found that the ash of the milk and of the new-born animal were nearly identical; no such result was found in the case of the child. In a later paper Hugounenq[4] gives tables showing the results of the analysis of the ash of fetuses at all stages of gestation.

The effect of the ingestion of **alcohol on the blood of the mother and fetus and on the milk** has been studied by Nicloux.[5] A woman in labor was given rum; fetal blood from the placenta was found to contain a considerable quantity of alcohol. After the administration of even small doses of alcohol to a woman some of it could be found in the milk. In experiments upon animals (dog, sheep, etc.) the amounts of alcohol in the blood and milk of the mother and in the blood of the fetus were found to be approximately equal.

**Blood of Mother and Fetus.**—Bidone and Gardini[6] find that the number of corpuscles and the amount of hemoglobin in the mother's blood decreases during pregnancy, so that at the end of pregnancy the former are reduced by one-half million per cubic millimeter and the latter by one-fifth. In the fetus at term the hemoglobin is one-fifth greater than in the adult, and the corpuscles are more numerous by 1,500,000 per cubic millimeter. Blumreich[7] finds the alkalinity of the blood during pregnancy to be increased. In rabbits the alkalinity (by Loewy's method) corresponded to 0.45% NaOH (normal 0.33%);

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[1] Compt. rend., CXXX, p. 941.

[2] Jour. de Physiol., II, p. 1.

[3] Zeit. f. Biol., XXXIX, p. 173.

[4] Jour. de Physiol., II, p. 509.

[5] Compt. rend. de la Soc. de Biol., Dec. 22, 1899; and Compt. rend. l'Acad. de Sci., CXXX, p. 855.

[6] Arch. ital. de Biol., XXXII, p. 36.

[7] Arch. f. Gynäk., XLIX, p. 699.

in pregnant women there was a distinct but less marked increase in the alkalinity.

Grandis [1] states that 33.46 % of the **ash of the placenta** consists of phosphoric acid—an extraordinarily large amount when it is remembered that even bone contains only 36.37 % and blood but 13.2 %. The percentage is even larger when the blood has been washed out of the vessels. Calcium is also present in large amount. Sodium is more abundant than potassium ; in this respect the placenta resembles blood-serum rather than most organs.

**The Florence test for semen** (the formation of characteristic crystals when a solution of I in KI is added to semen) has been thought by some recent writers to be due to cholin or lecithin. Vertun [2] has investigated the entire subject, and reaches the following conclusions : Semen which is allowed to stand for some time contains cholin ; such semen gives an especially well-marked Florence reaction. On the other hand, fresh semen from which cholin and similar bases were shown to be absent also showed the reaction ; hence cholin can not be the only substance in semen giving it. Besides the cholin group (cholin, muscarin, and neurin) the following basic substances give the reaction : spermin, adenin, xanthin, hypoxanthin, guanin, and carnin. It is evident that the reaction can not be considered at all conclusive as to the presence of semen. Vertun also discusses the value of negative results : *i. e.*, if the reaction is not obtained, is a person justified in stating that semen is not present ? Such a negative result has but little value, for there are many substances (small quantities of blood, urine, etc.) which interfere with the reaction. The reaction is useful in those cases in which the bodies other than spermin giving it can reasonably be supposed to be absent (as is often the case) ; but a careful search should always be made for the spermatozoa.

[1] Centralbl. f. Physiol., XIV, p. 115 ; from Atti Accad. d. Linc. (5), IX, 7, p. 262.
[2] Centralbl. f. Harn u. Sexualorgane, XI, p. 1.

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[See *American Text-Book of Diseases of the Eye, Ear, Nose, and Throat*, page 3.]

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